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I.

UPON THE PRESENT STATUS OF OTOSCLEROSIS.*

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The publications of the year just past, and especially the therapeutic suggestions which have been made, show us that we have by no means become united upon the conception of otosclerosis.

Before I enter into the discussion of this theme, it seems necessary for me to state clearly what I and my German colleagues understand by the term otosclerosis.

In my article on stapes ankylosis, which I read in 1903 before the meeting of the German Otological Society at Wiesbaden, and in my monograph on otosclerosis, I have set down for the conception of this disease the following definition:

We understand by otosclerosis a disease in which there is a permeable tube and a normal tympanic membrane, accompanied by a definite and characteristically marked clinical picture of a progressive difficulty in hearing, as shown by the functional tests. As a pathologicoanatomic foundation for the disease, investigations have revealed a loss of movement of the stapes, brought on by bony ankylosis in its framework or in the niche

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of the oval window, and a progressive spongification of the bony capsule of the labyrinth.

To this definition, upon the ground of our present knowledge, must be added that in addition to the disease of the bone, there is to be found, as shown by a histologic examination of a large number of cases, an atrophic degenerative process in the nerve endings in the membranous labyrinth.

Only those forms of disease which clinically and pathologico-anatomically correspond to the above definition will be dealt with, and not those forms of chronic affections accompanied by thickening of the interstitial connective tissue, which because of changes on the tympanic membrane are recognized as chronic adhesive processes or as the residues of long standing middle ear suppurations.

I understand thoroughly that the expression otosclerosis, which originated with v. Tröltsch, in no way corresponds to the pathologicoanatomic character of the disease. v. Tröltsch selected this name because he supposed that the otosclerosis led to an increased rigidity of the soft covering of the tympanic membrane, the hammer-anvil articulation, and especially of the window, which eventually led to a complete rigidity of the joint and to immobility of the stapes, in consequence of a calcification and ossification of the ligamentum annulare. He added, however, that anatomic examination would result in an increase in our knowledge of otosclerosis that would create for it a distinct position among ear affections. That this in reality is the case has been shown by the extensive literature in the last two decades describing the essential changes in the labyrinth capsule and the stapediovestibular articulation. We now know that lesions occur in the labyrinth capsule, in the ligamentum annulare, and in the footplate of the stapes which result in a clinical picture greatly resembling the symptoms appearing in middle ear disease. In a number of cases they absolutely correspond with the characteristic functional symptoms of middle ear disease, without demonstrable inflammatory changes in the soft coverings of the middle ear.

It has been repeatedly sought to substitute other expressions for otosclerosis. It has been proposed to designate the disease as "stapedioankylosis, with or without spongification of the labyrinth capsule." Heimann, in his article reported at Budapest, recommended the term "periostitis ossificans stapedio-

vestibularis," and Manasse in his recent monograph calls this disease process "otitis chronica metaplastica of the labyrinth capsule." Apart from the fact that the expression "periostitis ossificans stapediovestibularis" does not include those cases in which the disease process surely arises primarily from the bony labyrinth capsule, I hold it as more correct to cling to the term otosclerosis until we are entirely clear as to its etiology and pathologic anatomy; and this, even to this day, in spite of the countless and careful works upon the subject, is still unknown.

Upon the grounds already mentioned, and in order to prevent the proposal of other therapeutic remedies which are beneficial in the treatment of chronic adhesive processes or the remains of a chronic middle ear suppuration, but which can have no feasible influence on a process affecting the labyrinth capsule or the stapediovestibular joint, I desire to propose that this German conception of otosclerosis be universally accepted.

In regard to the etiology of the pathologic process in the labyrinth capsule and the stapediovestibular articulation, there are two opposite opinions: some authors hold the opinion that the changes are to be considered as secondary ones, that they are caused by an inflammation of the membrane lining of the middle ear. Habermann particularly holds this view, upon the ground of thirteen cases which he examined, the results of which caused him to believe that the bone disease comes from an inflammation of the middle ear, whence it is transmitted along the larger vessels to the bone, and is disseminated by means of the medullary spaces. Katz considers, as does also Toynbee for some of his cases, the disease to be a definite inflammation of the stapediovestibular articulation, upon a rheumatic basis, and designates it as "arthritis rheumatica." A similar opinion is held by Schilling, who says that in his case he observed a spongification of the labyrinth capsule which took its origin from the periosteum of the middle ear. Scheibe also contends that in those cases in which an earlier middle ear suppuration has been demonstrated, but only for those, a causative relation between the two diseases is possible.

In contrast to this conception of an inflammatory middle ear disease as the antecedent cause, most of the remaining authors who have made histologic examinations contend that otosclerosis is a disease appearing primarily in the bone. Moss in 1867 was the first to express the opinion that in view of the lack of

any change in the middle ear mucous membrane, the disease must be an ostitic process of the temporal bone.

Bezold and Scheibe, upon the ground of their histologic examinations, arrived at the belief that the bone disease took its origin either from the labyrinth capsule itself or from the periosteum of the niche of the oval window. The opinion that it is a primary bone disease of the labyrinth capsule is very energetically advanced by Politzer, who bases his views chiefly on the facts that in his large material most cases failed completely to show changes in the mucous membrane of the middle ear, and that the greatest changes were found in the deepest layers of the bone. This opinion is also held by J. Möller.

The pathologic anatomist Hanau, who examined the specimens from the cases published by Hartmann from the Siebenmann clinic, contends that the process at the stapes and the niche of the oval window is a hyperostosis with a rebuilding of the bone, and designates this newformed bone as metaplastic connective tissue bone, originating from the periosteum. Siebenmann, as the result of his investigations, concludes that the spongification does not arise from the periosteum, nor does it develop primarily from the labyrinth capsule, but that the oldest parts are to be found at the junction of the labyrinth capsule, which is formed out of the endochondrial substance, with the connective tissue bone arising secondarily from the periosteum (probably in the last named structure itself). Brühl also is of the opinion that we have to deal with a primary bone disease, which develops and progresses especially at the border between the endochondrial and connective tissue bone.

Hegener expresses as his opinion that in otosclerosis two distinct processes exist side by side. The bony change and the disease of the auditory nerve must be differentiated from each other, and the acoustic affection should not be considered as the result of the bony changes.

Manasse, in his monograph recently published, agreed with the opinion of Politzer and Siebenmann, that the pathologic changes which determine the clinical picture of otosclerosis are the product of primary disease of the labyrinth capsule. As to my own standpoint concerning the question of the point of origin of the pathologic changes in otosclerosis, Heimann and Fröschels, in their review, incorrectly stated that I consider otosclerosis a disease following a middle ear inflammation.

This is absolutely not the case. I have indeed granted the possibility that in those cases of otosclerosis accompanied by inflammatory changes in the mucous membrane of the middle ear, the disease might have taken its origin from the mucosa. But I further distinctly stated that this conception of the etiology of the disease leaves entirely unexplained the origin of the bone process in the labyrinth capsule in all cases in which a normal middle ear mucous membrane is demonstrated.

For these cases, only a primary disease of the bone or the periosteum can enter into consideration. That the bone changes can occur without cooperation of the periosteum, we are justified in believing, following the publications of Siebenmann, Politzer and Manasse, who found isolated foci in the cochlea which nowhere involved the middle ear mucous membrane. It is therefore certain that in a number of cases the pathologic changes take their origin absolutely from the bony labyrinth, while the middle ear mucous membrane cannot with the same certainty be designated as the originating point. It cannot, however, be excluded that in some cases the disease process extends from the mucosa of the middle ear into the bone; but it is just as possible that the mucous membrane changes found in otosclerosis are accidental, and it is not necessary to bring it into consideration as the originating point of the disease.

There prevails also no unity in the interpretation of the bone alteration. One group of authors holds the inflammatory character of the change as proven. Manasse especially holds this opinion, and upon the ground of his histologic investigations makes the following statement: "The commencement with the formation of osteoid and granulation tissue, the interchange between apposition, resorption and renewed apposition, the giant cell formation, these are all processes such as we find in chronic inflammatory processes in the bone—for example, in ostitis chronica fibrosa. There also occur the conversion of richly cellular marrow into fibrous marrow, the production of a hard, compact bone, and exostosis formation, all characteristics of a productive process analogous to chronic inflammation in other organs." Manasse proposed, therefore, for the entire disease process in the bone the name "ostitis chronica," and since the characteristic feature of this inflammatory process is the change in the old labyrinth capsule, he called the entire

affection "ostitis chronica metaplastica of the labyrinth capsule."

Brühl designates the bone changes in the labyrinth capsule, in agreement with Orth, as a spongy hyperostosis, and explains their origin as follows: By means of the vessels of the hyperplastic periosteum, and at the same time of the vessels of the Haversian canals of the periosteal built bone, a dissolving of the old bone is brought about, which is followed by a regular, even, excessive formation of connective tissue bone.

Siebenmann comes to another conclusion, after careful study of a large number of temporal bones. According to him, the beginning of the progressive spongification of the labyrinth capsule occurs not, as Manasse supposes, in an extension through new bone, but in the inward growth of cells from the periosteal tissue through the bone covering into the normal Haversian canals of the labyrinth capsule. Lacunar resorption from the walls, with the aid of mono- and polynuclear osteoblasts, goes hand in hand with it and fails in no case. New bone apposition results only where resorption processes have taken place. The newly apposed bone contains more chalk than the old bone, and does not deserve to be known as osteoid substance. As a differentiation from ostitis fibrosa, the spongifying process of the labyrinth capsule occurs without inflammatory symptoms. Granulation tissue, leucocytes and plasma cells are not present.

Since our colleague, Dr. Siebenmann, intends to demonstrate his newest specimens after my address, you will have the opportunity to judge for yourselves the correctness of the Siebenmann theory. But when we have arrived at a decision concerning its inflammatory or noninflammatory character, there remains still the questions: What is the essential cause? What gives the impetus to these alterations in the labyrinth capsule? Concerning this, also, there have been many hypotheses suggested. For a long time the authors have observed that otosclerosis is a disease which occurs more frequently in women than in men. Although Walb, as a reason for this phenomenon, suggests that the affection prefers the femal sex because in them is observed more frequently bad conditions of the blood and distinct anemia than in the male sex, which conditions he claims are the etiologic factors in otosclerosis, it is more probable that pregnancy and the puerperium are answerable for

this striking property of the sex. This opinion has much more value, when we remember that a large portion of these otosclerotic women trace the beginning of their difficulty in hearing to the time of pregnancy or the following period of convalescence.

The coincidence of pregnancy with the beginning of the otosclerosis suggests a causal relationship between the function of the hypophysis cerebri and the ear disease. It is well known that the hypophysis regularly enlarges during pregnancy and returns again to normal during lactation. We suppose that the hypophyseal changes during pregnancy depend upon a diminished function of the ovary. This conclusion is reached because the same hyperplasia of the hypophysis occurs in experimentally castrated female and male animals.

Furthermore, it has been possible to prevent or to limit the hypophyseal hyperplasia by feeding with extract of the ductless glands. We know, furthermore, that as a result of increase or disturbance of the internal secretion of the hypophysis, we find bony changes which are called acromegaly. In this disease are found exostoses and irregular porosity of the external layer, whose histologic examination shows that this is not a specific pathologic process, but that apposition and resorption take place exactly in the same manner as in normal bony growth. The causal relationship between the anomaly of the hypophysis and acromegaly is shown by the fact that the latter can be cured by operation for hypophyseal tumors.

Although the changes in the labyrinth capsule in otosclerosis are not analogous to the bony changes of acromegaly and the puerperal bony processes, still the fact that the bony changes for which the hypophysis is responsible and the beginning of otosclerosis both occur during pregnancy, gives food for thought, and I would like to call your attention to the possibility of an etiologic connection between malfunction of the hypophysis and the origin of otosclerosis, especially in persons with an hereditary taint. It would be a very good plan to examine cases of hypophyseal tumors, with or without acromegaly, for symptoms of otosclerosis. However, even if we are willing to concede that the cases of otosclerosis which begin at the time of pregnancy can be caused by lesion of glands with internal secretion, only a portion of the cases can be thus explained. Probably constitutional anomalies may cause the

disease. This view is supported by the fact that the disease is bilateral and the degree of deafness in many cases is the same on both sides. This similarity, in many cases, is represented on the pathologicoanatomic side by a complete symmetry of the bilateral bony changes, which caused Otto Mayer and Manasse to believe that a disease of the vascular system is the cause of the affection.

Undoubtedly, the most important etiologic factor is heredity. Although Panse could find in forty patients with synostosis of the stapediovestibular articulation only three with deaf brothers or sisters, Bezold, Denker and Siebenmann demonstrated in their cases of sclerosis percentages of 52, 40 and 35. The family trees of otosclerotics published by Körner and Hammerschlag are of special interest in the question of inheritance of the disease. Körner thought that not the disease itself but the predisposition to the disease was inherited, and believed that in otosclerosis it is always a case of inherited predisposition thereto. The explanation of the fact that we cannot in all cases show heredity connotes that the determinants representing the special peculiarity do not necessarily become active in each offspring, but can skip one or more generations and suddenly reappear. For these reasons, Körner agrees with Siebenmann that in otosclerosis there is an abnormal postembryonic developmental process.

Hammerschlag agrees with Körner that there is always an inherited predisposition in otosclerosis, but concludes that the primary appearance of otosclerosis is not to be sought among the lower mammals, but in the primates. He is convinced that if the disease ever appeared among the primates, a primary appearance among them is possible today. It is unnecessary to explain the apparently spontaneous appearance of cases of otosclerosis as cases of latent heredity. In his work, Hammerschlag gives two family trees in which both deafmutes and otosclerotics appear, and calls attention to the possibility of a genetic connection between these two aural lesions. As a basis for this, he states that in many cases of congenital deafness, the same bony lesions are found as in otosclerosis, and that in the latter degenerative atrophic changes in the membranous labryinth are frequently found which correspond to those found in congenital deafness. He agrees with Hegener that in otosclerosis there is not a secondary process in the acusticus, but

a primary one, coincident with and independent of the bone lesions. Often the disease in the nerve appears before that in the bone. Among other proofs is that of the subjective noises frequently appearing earlier.

On the grounds of his investigations upon deaf cretin children and on a deaf cretin dog, Alexander comes to the conclusion that otosclerosis is a congenital disease. Since the bony changes found in his cases completely agree histologically with those in the labyrinth capsule in otosclerosis, he is convinced that the latter disease is also a congenital one. He calls the lesion "ostitis vasculosa," and believes that the otosclerotic has very vascular bony foci in his temporal bones at his birth and even during his embryologic period, and that only at puberty, and in some cases much later, do they extend to the endosteum of the labyrinth or lead to fixation of the stapes, changes on the cochlear window and secondary degenerative processes in the nerve endings.

Brühl agrees with Alexander that the hereditary predisposition to the disease probably has its anatomic basis in a congenitally misplaced spongification focus in the labyrinth capsule, which begins to develop under the influence of certain stimuli. The changes which affect the vestibular articulations and their vicinity are probably due to a formative stimulation of bone and new formation consisting of pressure and traction which have their greatest action, on anatomic grounds, just at the anterior circumference of the vestibular window, the site of predilection for changes in stapes ankylosis. The interrupted traction on the bone caused by movement of the footplate of the stapes, sets up an irritation in the bone and periosteum. In addition, the continual contraction of the musculus tensor tympani, which likewise lies just at the anterior circumference of the stapes, causes a permanent unrest in the periosteum and bone of this region.

Brühl's hypothesis is opposed by O. Mayer. He believes that it is incomprehensible how the atypical cases (unconnected with the tympanic mucous membrane) in which stapes ankylosis is present should be due to traction on the annular ligament, and the formative stimulus should cause disease of the bone at remote places, while in the cases where there is no stapes ankylosis, the same foci should be due to some other cause. The fact that in many cases the lesions in the labyrinth capsule

show a complete symmetry, led Mayer to the belief that these foci correspond to blood vessels and are caused by their disease. The causes of such lesions seem to be above all disturbances in the vasomotor functions, but he thinks that other causes of local circulatory disturbances, especially organic disease of the blood vessels (arteriosclerosis), may cause the same changes. According to him, the heredity consists in an inheritance of the cause of the disease, for it is well known that heredity plays a great role in both arteriosclerosis and vasomotor neuroses. That Siebenmann considers otosclerosis to be a congenital condition, has already been noted above.

While the other authors could accept the heredity of otosclerosis only on the grounds of the history, and the subjective and objective findings, Manasse was able to show anatomically the typical bony changes in two members of the same family. There seems to be no doubt, therefore, that otosclerosis is actually an inheritable disease.

Of the other etiologic factors, lues, above all, has been accused of being the cause of the disease. Habermann made the most thorough study of this phase, and is convinced of the causal relationship between otosclerosis and syphilis. Later investigations have shown, however, that lues is not a frequent etiologic factor in producing the characteristic changes in the labyrinth capsule—first, because the vast dissemination of syphilis would cause a more frequent occurrence of the aural affection; and second, because men suffer from syphilis much more frequently than women, while otosclerosis is specially a disease of the female sex. But especially do the results of the Wassermann reaction argue against the correctness of Habermann's theory: the examination almost invariably gives results which speak against a luetic etiology.

Many authors (Panse, Hartmann, Siebenmann, etc.) have noticed that many patients refer their disease to a severe cold. Although we have no exact idea of the manner of action of low temperatures, we cannot reject the possibility of some such etiology, especially since very observant patients have noticed a permanent exacerbation of their trouble following considerable cooling off. Perhaps it is disturbance transmitted through the vascular system.

That a trauma may lead to stapes ankylosis with the functional symptoms of otosclerosis was shown by a case published

by Politzer in 1862. If we review what has been written concerning the etiology of otosclerosis, it seems correct to state that in the majority of cases, at least, we have to do with a hereditary anlage as the cause of the disease. This predisposition lays the foundation upon which, under the influence of certain stimuli, there arises the affection with its characteristic symptoms and pathologicoanatomic changes. These stimuli are the increased bony formation during puberty and the bony changes during pregnancy and the puerperium, which are probably dependent on the hyperplasia of the hypophysis, which appears during pregnancy. Furthermore, I believe in the correctness of Otto Mayer's claim, that disturbances in the circulation such as are present in arteriosclerosis, vasomotor neurosis, and lues may give the impetus to the development of the disease. It has not yet been proved that the continuous movement of the stapedial footplate and the permanent contraction of the musculus tensor tympani can be regarded as a cause for the fact that the bony alterations develop by predilection at the anterior circumferance of the vestibular window, as Brühl claims.

Shortness of the time at my disposal forbids me to go into an extended discussion of the pathologicoanatomic changes in the labyrinth capsule, the ligamentum annulare, and the stapedial plate. I must content myself with demonstrating the characteristic pictures with the projection apparatus, at the close.

As to the symptoms and the clinical course of the disease, I will be brief, since the greater part is well known. You know that frequently subjective noises of the most varied kinds (roaring, ringing, knocking, etc.) and of varying intensity are the first indications of the disease, and that these noises are sometimes so severe and act so unfavorably upon the patient, that they are regarded by them as a greater evil than the deafness. According to the statistics of Bezold, Politzer and myself, they are observed in 70 to 80 per cent of cases of otosclerosis.

The diminution of hearing usually appears very gradually, so that the patient sometimes cannot tell when it began. Often only one ear is affected, and the other not until later. The course is usually insidious, and frequently many years pass before the deafness becomes very great. However, frequently rapid exacerbations are seen. As to the degree of deafness and the results of exact functional tests, they are dependent

on the location and extent of the changes in the capsule, the annular ligament, the footplate and the membranous labyrinth. The variability of these lesions and the resulting symptom complex cause the disease to be divided into three forms:

(1) The pathologic changes are located exclusively at the vestibular window and its vicinity, and have led to a bony fixation at the footplate—isolated stapes ankylosis.

(2) Appearance of multiple spongifying foci in the labyrinth capsule, with atrophic degeneration of the membranous labyrinth without stapes ankylosis.

(3) Bony fixation of the stapes plate, combined with other foci in the labyrinth capsule and with atrophy of the membranous labyrinth.

If it is a case of purely stapes ankylosis (group 1), whisper is usually heard at a short distance from the ear. Numbers 9 and 5 (German) are heard the poorest. By an exact examination we can obtain the symptom complex known as Bezold's triad, characterized by raising of the lower tone limit, lengthening of the bone conduction in the Weber-Schwabach test, and negative Rinné. The upper tone limit is usually normal or very slightly lowered.

If there are isolated foci in the labyrinth capsule, and if an atrophic degeneration of the membranous labyrinth results from the extension of these foci to the endosteum of the cochlea, or from other causes (group 2), without changes in the stapediovestibular articulation, the functional symptoms of nerve deafness appear. Whisper is not heard in many cases, and loud tones are often heard only at a short distance. The perception is worst for words containing high sounds (6, 7). Although the lower tone limit can be about normal, the upper is greatly lowered. Bone conduction (Weber-Schwabach) is shortened and Rinné is positive.

In the combination of stapes ankylosis with spongifying foci in the labyrinth capsule and atrophy of the membranous labyrinth (group 3), the following functional symptoms are observed: Hearing distance for speech and whisper greatly reduced; the lower tone limit is raised with a simultaneous lowering of the upper; bone conduction is not increased and in many cases is decreased, while Rinné is not negative, but briefly positive and even normal. We have, therefore, functional symptoms which speak neither for an uncomplicated

affection of the conduction apparatus nor for a pure disease of the internal ear. In such cases we must use the other methods of examination, which will be mentioned in discussing the diagnosis.

Disturbances of equilibrium and attacks of dizziness of different degrees are frequently observed in otosclerosis. They may be referred to a direct or indirect change in the vestibular nerve or its endings in the vestibule and ampullæ. Siebenmann proposed the theory that pressure and density changes which the labyrinthine fluid undergoes through the influence of the spongification extending to the endosteum of the labyrinth, are responsible for the lesion of the function of the nervus cochlearis as well as disturbances of equilibrium. He thinks that the newformed spongiosa can break through into the perilymph, and by means of the sudden increase of intralabyrinthine pressure and changes of position cause Meniere's disease, both in its milder forms and in the form of apoplectic attacks of vomiting, nystagmus and falling. In the same way Siebenmann explains the sudden appearance of loud subjective noises, such as thunder, popping, shooting, etc.

Pain in the depths of the ear is relatively seldom observed in otosclerosis, and is to be referred to changes in the periosteum or the stapediovestibular articulation, or to changes in the sensory fibers of the nervus facialis. The patients comparatively frequently complain of a feeling of pressure or tension in the ear.

The hyperemia of the promontorial mucous membrane, which Schwartze claimed is the characteristic symptom of otosclerosis, has been observed both in nerve deafness (Siebenmann) and in normal hearing (Brühl), although rarely. These cases, however, may be initial symptoms of cases of otosclerosis, which later develop into a severer form, and we can regard promontorial hyperemia as a symptom which, if not absolutely certain, is at least one which, with others, aids in the diagnosis of otosclerosis.

The paracusis Willisii, so often observed in this disease, was explained by Willis himself as due to a slack drum. Toynbee explained it as a result of a shaking of the chain of ossicles, and Politzer also thought that the ossicles whose articulations had become rigid had by this shaking been displaced from their fixed positions and thus made suitable for conducting

sound. Siebenmann gives the following explanation. He claims that the otosclerotic is disturbed less by street noises, such as railway journeys, than the normal hearing individual, because they are composed of lower tones, the perception of which he has lost. To this is added the fact that normal hearing people speak louder in loud noises, and for this reason the otosclerotic can apparently hear loud sounds better than in quiet places. Urbantschitsch explains the paracusis Willisii by an increased function of the nervus acusticus.

A symptom observed frequently by patients with nerve deafness as well as by otosclerotics, to which Urbantschitsch and Walb have drawn attention, is the decrease in hearing, the rapid weariness following strained attention.

Recently Fröschels described a symptom consisting of a diminution of the tickling sensation parallel to the loss of hearing. If a small probe armed with a cotton brush is introduced about 1 cm. into the meatus and moved gently to and fro, it will often be found that the tickling sensation is less developed in the worse ear. In this test it is of course understood that no suppuration exists or has existed, since this also disturbs the sensibility.

The diagnosis of otosclerosis is easy in those cases where the process is confined to the stapediovestibular articulation and its vicinity (group 1). If the drum is normal, and if catheterization does not improve the hearing, while showing the permeability of the tube, we can say that it is a case of stapes ankylosis due to otosclerosis, when we find a distinct loss of hearing and the symptom complex known as Bezold's triad (raising of the lower tone limit, lengthening of bone conduction, negative Rinné). The cases where the diagnosis was made on these findings invariably showed its correctness whenever they came to postmortem.

The diagnosis is more difficult when the disease is not confined to the vicinity of the vestibular window, but affects the whole labyrinth capsule to a greater or less extent (group 2). If there is not complete or a high degree of deafness due to degenerative atrophy in the labyrinth, we can usually make the differential diagnosis between nerve deafness and a combination of it with stapes ankylosis. For this we use the test described by Gelle. If the Rinné is positive, and there is a marked raising of the lower tone limit, and if Gelle's test is

negative—if compression of air in the meatus has no effect upon the intensity of the tuning fork's note—it is very probable it is a case of otosclerosis, i. e., stapes ankylosis combined with extensive spongification of the labyrinth capsule and atrophy of the membranous labyrinth.

In addition to the above, we can use in such cases a test described by me. If doubtful cases are tested with a continuous series of tuning forks as to the duration of their perception, it will be possible to demonstrate in otosclerosis that the part of the scale still heard will have its notes heard by air conduction longer in proportion to the number of vibrations, which is not the case in pure nerve deafness. If by this test we obtain a curve which in general is an ascending one and shows only unimportant deviations, we can with almost complete confidence make the diagnosis of stapes ankylosis complicated by an involvement of the internal ear. If, however, the test gives a very irregular line, it is an exclusively labyrinthine affection, i. e., a purely nerve deafness.

On account of what has been written, I cannot share the skepticism of Panse and Manasse, who believe it is impossible in very many cases to make the diagnosis of otosclerosis. It must of course be admitted that the small isolated bone foci in the labyrinth capsule cannot be found intra vitam, and that extensive disease of the labyrinth capsule without stapes ankylosis cannot be diagnosed from nerve deafness by functional tests. The other cases, however, can be confirmed by the functional tests, bearing in mind the anamnesis and objective findings. It must be remembered that most cases of otosclerosis are found between the ages of twenty and forty, while nerve deafness usually does not appear until after forty.

In the treatment of otosclerosis, this prophylactic consideration must be remembered. Since experience has taught us that exacerbations of otosclerosis follow pregnancy and the puerperium, the prevention of conception is strongly urged. Furthermore, consangineous marriages are to be forbidden, since the offspring are often deaf or very hard of hearing. They are especially to be forbidden if the parents already show a distinct taint of otosclerosis. The otosclerotic, furthermore, must beware of "catching cold," although we cannot explain just how a rapid cooling off of the body has any influence on changes in the labyrinth capsule. Probably it is an influence

acting through the vascular system. Finally, the otosclerotic must beware of cold douches upon the head, and sea voyages, since experience has shown that the disease is made worse thereby. As to the active treatment of manifest otosclerosis, I regret to say that I have the same viewpoint today that I expressed in my monograph. It is, that because of the kind of osteopathologic process, a therapy based on mechanical action or local operative treatment will have only a slight effect or none at all. This kind of treatment is justified in chronic adhesive processes and in the results of chronic suppuration of the middle ear, but not in otosclerosis.

All treatments directed toward mobilizing or extracting the stapes have resulted in so small a permanent improvement in the hearing that they are not to be recommended, especially since there is a possibility that they may make the disease worse.

When there are subjective noises, we have seen more or less permanent result in some cases from the use of the electric drum masseur, after catheterization and pressure probing proved useless. Since these subjective noises are often especially troublesome in otosclerosis, I think we are justified in using this massage treatment repeatedly, even when the result obtained is only transitory.

For internal treatments, Politzer prescribes potassium iodid in doses of 1 gram daily, and he believes that it is possible in some cases to check thereby the further progress of the case. He recommends treatments of ten to fifteen days, in intervals of two months, four or five times a year.

The favorable results which Vulpius, Brühl, Alt and Eitelberg obtained by thyroid treatment have not been confirmed by others.

I have had no experience with the transtympanal electroionization recommended by Malherbe, but I cannot conceive how such a treatment would have any effect on a bone process in the labyrinth capsule. It seems to me to be doubtful also whether the results which Malutin claimed he obtained with mud baths will be confirmed by others. Furthermore, the fibrolysin treatment, with which Erbstein obtained a slight improvement in the hearing of one out of four cases, does not seem to me to offer much hope. Whether the diplococcus serum recommended by Ferreri can have any effect in the dis-

ease of the labyrinth seems to me very questionable. The experiments of Müller with the serum antisclereux Malherbe had no special result.

More is to be expected, according to my experience, from the internal administration of phosphorus, as recommended by Siebenmann. Upon the basis of experiments by Mirwa and Stotzner, which showed that phosphorus, when rationally used, is able to prevent the formation of the normal spongiosa, at least in long bones, and to favor the production of compact bone, Siebenmann gives daily one to two tablespoonfuls of Kassowitz's phosphorus emulsion, to check the spongifying process in the labyrinth capsule. For many years I have in my clinic treated otosclerosis with phosphorus, for the last three years in the form of phytin (phytin 0.25 in original tablets, two or three daily, after meals), which is well borne in most cases. Although we cannot expect too much from this therapy, still I think we can give patients the hope that the process will be checked and that the subjective noises will be favorably influenced, perhaps made to disappear. Of course, we cannot be sure whether the improvement is not to be often referred to a favorable influence of the drug upon the general condition of the body and of the nervous system.

As I stated above, it seems to me that there is a possibility of a causal relation between otosclerosis and improper function of the hypophysis. For this reason, in my clinic we have begun to prescribe hypophysin or pituitrin, especially since Moraczewski's experiments have shown that feeding with hypophysin tablets prevents the elimination of phosphorus. According to v. Fränkel-Hochwart and Frölich, the hypophyseal preparation is practically innocuous. The hypophysin is prescribed in tablets (two or three daily).

Since the enlargement of the hypophysis which regularly occurs during pregnancy has a possibly causal connection with the origin or exacerbation of otosclerosis, which is often noted during this period, we may try in the case of women with hereditary taint to prevent the hypophyseal hypertrophy or to restrict it by exhibition of extract of sexual glands. That this can be done, has been noted above. We prescribe ovarian tablets (Merck) or spermin tablets (Poehl), two or three daily.

As to otosclerol, which has recently been recommended, and

which has yielded good results as far as tinnitus is concerned, I have not had sufficient experience. Likewise, I have not had sufficient opportunity to test the value of vasotonin, which has very recently been recommended for otosclerosis.

If the disease is bilateral, and the hearing is greatly reduced, so that intercourse with people is greatly hindered, it is necessary strongly to urge the patient to take a course in lip reading, in order that the eyes may at least partially assist the hearing.

(Demonstrations of photograms of specimens of otosclerosis (Katz, Bezold-Scheibe, Politzer, Siebenmann, Habermann, Brühl) with the projectoscope.)

II.

THE REPORT OF AN EXAMINATION OF BOTH TEMPORAL BONES FROM A HUNDRED AND TWENTY INDIVIDUALS IN REFERENCE TO THE QUESTION OF SYMMETRY IN HEALTH AND DISEASE.

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PREFATORY NOTE

The examination was undertaken to ascertain facts as to the symmetry of the temporal bones, a subject on which little reference is made in otological and anatomical literature, but which has, I venture to think important bearings on the causation, diagnosis and treatment of disease.

The following is a list of the sex and age of the individuals:

1.	M.	$\frac{9}{12}$.	*22.* F. 29.	*43.* F. 38.
2.	F.	$1\frac{7}{12}$.	*23.* M. 30.	*44.* M. 39.
3.	?.	$1\frac{9}{12}$.	*24.* M. 30.	*45.* M. 39.
4.	M.	2.	*25.* M. 30.	*46.* M. 39.
5.	F.	2.	*26.* F. 30.	*47.* M. 39.
6.	?.	$2\frac{2}{12}$.	*27.* M. 33.	*48.* M. 39.
7.	M.	$3\frac{3}{12}$.	*28.* M. 33.	*49.* F. 41.
8.	F.	6.	*29.* M. 33.	*50.* M. 42.
9.	M.	9.	*30.* F. 33.	*51.* F. 42.
10.	M.	11.	*31.* M. 34.	*52.* M. 43.
11.	F.	16.	*32.* M. 35.	*53.* F. 43.
12.	M.	20.	*33.* F. 35.	*54.* M. 44.
13.	F.	21.	*34.* M. 36.	*55.* M. 45.
14.	F.	22.	*35.* F. 36.	*56.* M. 45.
15.	M.	24.	*36.* F. 36.	*57.* M. 45.
16.	M.	24.	*37.* M. 37.	*58.* M. 47.
17.	F.	25.	*38.* M. 37.	*59.* M. 48.
18.	M.	28.	*39.* F. 37.	*60.* M. 48.
19.	M.	28.	*40.* M. 38.	*61.* F. 48.
20.	F.	28.	*41.* M. 38.	*62.* F. 48.
21.	F.	28.	*42.* F. 38.	*63.* M. 49.

64. M. 49.	*83.* M. 57.	*102.* M. 64.
65. M. 49.	*84.* F. 57.	*103.* F. 64.
66. F. 49.	*85.* M. 58.	*104.* F. 64.
67. M. 50.	*86.* M. 58.	*105.* M. 66.
68. F. 50.	*87.* M. 59.	*106.* F. 66.
69. F. 50.	*88.* M. 59.	*107.* M. 67.
70. M. 51.	*89.* M. 59.	*108.* M. 68.
71. M. 51.	*90.* M. 59.	*109.* M. 68.
72. M. 51.	*91.* M. 60.	*110.* M. 71.
73. M. 52.	*92.* M. 60.	*111.* M. 72.
74. M. 53.	*93.* M. 61.	*112.* M. 72.
75. F. 53.	*94.* M. 61.	*113.* F. 72.
76. F. 54.	*95.* F. 61.	*114.* F. 73.
77. F. 54.	*96.* F. 61.	*115.* F. 76.
78. F. 55.	*97.* F. 61.	*116.* F. 77.
79. F. 55.	*98.* F. 62.	*117.* F. 81.
80. F. 55.	*99.* F. 62.	*118.* M. 82.
81. M. 56.	*100.* F. 63.	*119.* F. 86.
82. F. 56.	*101.* M. 64.	*120.* F. 89.

I regret the deficiency in the number of infants and children, but this part of my collection is still in its infancy. The sex is known in all except in two, one aged one year and nine months and the other two years and two months. There are sixty-eight males and fifty females. The specimens are arranged according to age, and if there are several of the same age, the males are placed before the females. Each bone is separately placed and numbered, the right immediately preceding its corresponding left bone; the odd numbers therefore represent the right and the even numbers the left bones.

In referring to specimens the following method is used:

$\frac{\text{Number of the right bone}}{\text{Number of the left bone}}$, sex, age; for example, $\frac{11}{12}$ F. 36.

The bones are, in the main, vertically sectioned through the outer antral wall, mastoid process, middle-ear tract and labyrinth. Sections in other directions have been made when thought necessary. Further sections will be made immediately if any members of the Congress express such a wish.

The report is considered under two main headings: (A) In health. (B) In disease.

A. In Health

This part is considered under two headings: (1) The Exterior; (2) the Interior.

1. The Exterior

(*i*) *The external surface*

Symmetry is the rule in markings, shape and size.

(*a*) In one specimen *the temporal ridge* behind the antrum is more prominent on the right side, $\frac{1}{1}\frac{1}{1}$ M. 56.

(*b*) In two sets the *posterior zygomatic line* on the right side above the antrum has an inclination downwards and backwards and an overhang ng appearance associated with a markedly high-lying antrum, conditions which are not seen on the left side and in which the antrum is not high lying, $\frac{1}{1}\frac{1}{1}$ M. 53 and in $\frac{1}{1}\frac{1}{1}$ M. 61.

(*c*) *A specially large foramen*, evidently from a vessel, is present immediately above the meatal spine, leading to the coarse cells in the outer antral wall on both sides in $\frac{1}{1}\frac{1}{1}$ M. 57.

(*d*) *Perforations in the cortex of the supra-meatal triangle* exposing the cells lining the outer antral wall are present, on the right side only in $\frac{1}{1}$ M. 38 and on the left side only in $\frac{1}{1}$ F. 25. There is no evidence of disease in either.

(*e*) *The supra-meatal spine* presents many variations and one side is occasionally larger than the other, but symmetry is the rule.

(*f*) The remains of the *masto-squamosal suture* when present are usually symmetrical, but in three sets one side is more marked than in the other; one on the right side, $\frac{1}{1}\frac{1}{1}$ F. 64, and two on the left, $\frac{1}{1}$ M. 24, $\frac{1}{1}\frac{1}{1}$ F. 66.

(*g*) There is very little *variation in the size and shape of the mastoid process*; the left is slightly larger and more pronounced in $\frac{1}{0}$ M. 2 and the right in $\frac{1}{1}\frac{1}{1}$ M. 61 and $\frac{1}{1}$ F. 41, where the right cellular mastoid is rounder than the diploëtic

left. Even in sets which vary strikingly in their interior there is no striking external variation, as in $\frac{1}{1}\frac{4}{3}\frac{3}{3}$ F. 56, $\frac{1}{1}\frac{3}{3}\frac{3}{4}$ F. 61, $\frac{2}{2}\frac{3}{2}\frac{7}{3}$ F. 73, in all of which one bone is cellular and the other is diploëtic.

In connection with this, Iwanoff (Politzer's text-book, page 39) states that in brachycephalics the right mastoid process is poorly developed.

(*h*) *Thinning and perforation of the anterior surface of a cellular mastoid process*, close to the tympanic plate, is seen on the left side in $\frac{1}{1}\frac{1}{8}$ M. 48. The condition is not due to disease in the mastoid cells, and would have passed without comment if precisely the same thing had not been seen in the left bone of a man, age forty-five, not appearing in this series.

(*i*) *A large and unusually situated external opening for the petro-squamosal sinus* between the base of the zygoma and the post-gleniod tubercle is seen on the right side only in $\frac{1}{1}$ M. 9.

(*j*) The variations in the opening of the mastoid emissary vein will be considered when dealing with the posterior surface.

(*ii*) *The superior surface*

(*a*) *Grooving for the petro-squamosal sinus* is very well marked in three sets, although remains may be seen in others. There may be marked asymmetry.

$\frac{1}{2}$ M. $\frac{0}{1}\frac{0}{2}$, the grooving is seen at the back part, that on the left side being more marked.

$\frac{1}{1}\frac{3}{4}$ M. $3\frac{0}{1}\frac{0}{2}$, the grooving is well marked on both sides, but is larger and deeper on the left. There is an external opening on both sides, the left being the larger, in the most usual position between the post-glenoid tubercle and the tympanic plate. On the right side the sinus passes under a bridge of bone before opeing into the lateral sinus, while on the left side it is an open groove all the way.

$\frac{1}{1}\frac{7}{8}$ M. 9, the grooving is large and deep all along the line of suture on the right side, the sinus passing under a bridge of bone behind to open into the lateral sinus groove; in front it perforates by a large opening between the base of the zygoma

and the post-glenoid tubercle—a rare position. On the left side there is no grooving, the only existing sign being an external opening at the outer extremity of the Glaserian fissure.

$\frac{121}{122}$ F. 48, the grooving is present on the left side only. there is no external opening.

(*b*) *Dipping down of the middle fossa,* altering the shape of the antrum by depressing its roof or caus ng the dura mater to intervene between the upper part of the cavity and the outer surface, occurs in nineteen sets. It is never symmetrical. It is present on both sides in six, but always more on one side than the other.

On both sides, but more marked on the right:

1. $\frac{107}{116}$ F. 57.
2. $\frac{123}{134}$ F. 61.
3. $\frac{208}{210}$ M. 66.
4. $\frac{227}{226}$ F. 73.

On both sides, but more on the left:

5 $\frac{188}{200}$ F. 63.
6. $\frac{203}{204}$ M. 64.

On the right side only in six:

1. $\frac{33}{34}$ F. 25.
2. $\frac{27}{28}$ M. 28.
3. $\frac{14}{15}$ M. 30.
4. $\frac{61}{52}$ F. 30.
5. $\frac{111}{112}$ M. 45.
6. $\frac{163}{164}$ F. 56.

On the left side only in seven:

1. $\frac{13}{14}$ M. 37.
2. $\frac{83}{84}$ M. 39.
3. $\frac{121}{122}$ F. 48. Depression behind the antrum.
4. $\frac{161}{162}$ M. 56.
5. $\frac{187}{188}$ M. 61.
6. $\frac{189}{190}$ F. 61.
7. $\frac{211}{212}$ F. 77.

It will be seen that it occurs about equally in the two sexes, nine being males and ten females.

It cannot be stated that the type of bone as regards presence or absence of cells has any influence in its production, for it is seen in all types. There is no external sign of its presence apart from cases of hydrocephalus which is not here represented.

The antrum may be high lying and yet the middle fossa may dip down, shutting part of the cavity out from the surface, as on the right side of $\frac{1}{11}$ M. 45.

(*c*) *A deep groove is present in the neighbourhood of the hiatus Fallopii on* the left side only in $\frac{1}{11}$ M. 39, and on the right side only in $\frac{1}{111}$ F. 81; it is apparently for a vessel.

(*iii*) *The anterior surface.*

(*a*) *The openings for the petro-squamosal sinus* have already been alluded to.

(*b*) *The opening in the tympanic plate* is closed by bone on both sides at an early age in $\frac{0}{10}$ F. 2. On the other hand they are both unclosed by bone in $\frac{1}{16}$ F. 21 and $\frac{1}{11}$ F. 38. In $\frac{1}{11}$ M. $3\frac{3}{12}$ the opening is closed on the left side and not on the right.

(*c*) Sometimes, by no means always, above the age of sixty years *the tympanic plate undergoes marked thinning*, and a gap closed by membrane only appears in the atrophied bone, closely resembling the condition seen in early life. It is apparently due to movements of the jaw. It may be seen on both sides in—

$\frac{2}{2}\frac{2}{2}\frac{5}{6}$ F. 72.
$\frac{2}{2}\frac{3}{3}\frac{7}{5}$ F. 86.

On the right side in—
$\frac{2}{2}\frac{3}{3}\frac{5}{6}$ M. 82.

On the left side in—
$\frac{2}{2}\frac{0}{0}\frac{5}{6}$ F. 64.

(*d*) *Bony spicules crossing the floor at the bend of the internal carotid canal* are seen on the left side of $\frac{1}{11}$ M. 24. They are not present on the right side, but there is a small groove separated from the canal floor by a slight ridge.

(*iv*) *The posterior surface*

(*a*) *The lateral sinus.* As is well known, the right lateral sinus is usually larger and more forward than the left whatever the type of bone, owing to the opening of the longitudinal sinus being usually towards that side, and as Politzer has pointed out, the sinus is more forward in diploëtic and slightly cellular bones, which points are borne out by this series. Iwanoff, previously referred to, states that in brachycephalic skulls the right temporal bone is not so well developed as the left and the right lateral sinus is far forwards and outwards. On this question I am not in a position to judge.

The left lateral sinus groove is, however, not infrequently, the larger, for there are twenty-six instances, a proportion much larger than one expected. It is more frequent in males than females.

1. $\frac{11}{12}$ $2\frac{2}{12}$.
2. $\frac{13}{14}$ M. $3\frac{3}{14}$.
3. $\frac{28}{28}$ F. 28.
4. $\frac{25}{26}$ M. 30.
5. $\frac{55}{66}$ M. 33.
6. $\frac{68}{76}$ F. 36.
7. $\frac{71}{72}$ F. 36.
8. $\frac{73}{74}$ M. 37.
9. $\frac{78}{88}$ M. 38.
10. $\frac{85}{86}$ F. 38.
11. $\frac{87}{88}$ M. 39.
12. $\frac{111}{112}$ M. 45.
13. $\frac{116}{118}$ M. 47.
14. $\frac{137}{138}$ F. 50.
15. $\frac{139}{140}$ M. 51.
16. $\frac{153}{154}$ F. 54.
17. $\frac{165}{166}$ M. 57.
18. $\frac{167}{168}$ F. 57.
19. $\frac{173}{174}$ M. 59.
20. $\frac{175}{176}$ M. 59.
21. $\frac{177}{178}$ M. 59.
22. $\frac{185}{186}$ M. 61.
23. $\frac{194}{196}$ F. 62.
24. $\frac{213}{214}$ M. 67.
25. $\frac{217}{218}$ M. 68.
26. $\frac{239}{240}$ F. 89.

Males	16
Females	9
Sex unknown	1
Total	26

The greatest discrepancy in size of the grooves is seen in—

$\frac{111}{112}$ M. 45, where the left is three times the size of the right.

$\frac{177}{178}$ M. 53, where the left is very small.

$\frac{153}{154}$ F. 54, where the right is very small.

$\frac{185}{186}$ M. 61, where the left is very much larger than the right.

Equality in size and forwardness is seen in—

$\frac{41}{42}$ F. 28 Both grooves are large and both bones are mostly diploëtic.

$\frac{107}{108}$ M. 44. Both grooves are large and both bones diploëtic.

$\frac{145}{146}$ M. 52. Both grooves are large, the right bone being cellular and the left partly cellular and partly diploëtic.

The larger sinus is not always the more forward, for in the bilateral diploëtic and slightly cellular types both sinuses may be equally well forward, even though differing in size.

$\frac{101}{102}$ F. 37, where both sinuses are large and equally well forward, the right being somewhat the larger. Both bones are diploëtic.

$\frac{111}{112}$ M. 45, where the right sinus is one third the size of the left and yet both are equally forward. The right bone is almost entirely diploëtic and the left is cellular except for the lower mastoid, which is diploëtic.

Extreme forwardness, the sinus reaching the posterior meatal wall, and being especially large in size, is seen in—

$\frac{108}{110}$ M. 45 on both sides, shutting out the apex of the antrum from the surface, both bones being diploëtic.

$\frac{131}{132}$ F. 54 on the right side, completely shutting out the antrum from the surface, the bone being diploëtic. The left bone is cellular.

$\frac{133}{134}$ M. 60 on the right side, shutting out the apex of a high lying antrum, the bone being diploëtic. The left bone is cellular.

Great forwardness in a cellular bone is seen in—

$\frac{38}{39}$ M. 39 on the right side, with a high lying antrum.

$\frac{215}{216}$ M. 82 on the right side.

The sinus is not always well forward in the diploëtic or slightly cellular types.

$\frac{27}{28}$ M. 28 where both bones are diploëtic and the right sinus is large and well forward and the left is not.

$\frac{28}{29}$ F. 29. Ditto.

$\frac{1}{1}\frac{1}{1}$ F. 50 where both bones are diploëtic and the left sinus is large and well forward and the right is not.

$\frac{1}{1}\frac{1}{1}$ F. 61, where the right bone is diploëtic and the left finely cellular throughout, neither sinus being well forward.

A deep pocket or sulcus in the left lateral sinus groove just before its exit is present in $\frac{1}{1}\frac{1}{1}$ M. 47.

(*b*) *The mastoid emissary vein.*—This vein usually leaves the lateral sinus at a corresponding point on the two sides; although that point may vary immensely, it is in the great majority of cases, below the knee of the vessel, for in one set only does it come off above the knee on both sides, the right vein being the larger and the left rudimentary:

$\frac{1}{1}\frac{1}{1}$ M. 39.

The internal opening in the bone is most frequently in the groove itself, or in its lower edge, but it may be some distance away, the vein running in an open groove before entering the bone, as on both sides of—

$\frac{1}{1}\frac{1}{1}$ M. 48,

or on one side only, as in—

$\frac{1}{1}\frac{1}{1}$ M. 59.

The point of emergence, which is subject to considerable variation in position, is usually symmetrical, but one may be further back than the other, as in—

$\frac{1}{1}$ M. $3\frac{3}{12}$, where the left is in the occipital suture and the right farther forwards.

One side may be higher than the other, one running upwards and the other downwards, as in—

$\frac{1}{1}\frac{1}{1}$ M. 53, where the right runs downwards and the left upwards.

A regular sulcus at the point of emergence is occasionally seen:

On the right side only in—

$\frac{1}{1}$ M. 9, with a diploic vein opening into it above, and,

$\frac{1}{1}\frac{1}{1}$ M. 49.

$\frac{1}{1}\frac{1}{1}$ F. 61, where the right starts double and emerges single.

The knowledge of the possible variations is important in cases of thrombosis of the lateral sinus when the vein is likely to be implicated and require dealing with.

(v) *The inferior surface*

(a) *The sulcus jugularis* is broadly speaking larger and higher lying on the side which has the larger lateral sinus namely the right and this is especially well marked in—

$\frac{1}{1}$ M. 28.
$\frac{1}{1}\frac{1}{1}$ M. 43.
$\frac{1}{1}\frac{1}{1}$ F. 48.
$\frac{1}{1}\frac{1}{1}$ M. 53.
$\frac{1}{1}\frac{1}{1}$ F. 66.

In 32 sets, however, the left sulcus is larger than the right.

1.	$\frac{1}{1}$	M. 2.	*12.*	$\frac{1}{1}$	F. 38.	*23.*	$\frac{1}{1}\frac{1}{1}$	M. 57.
2.	$\frac{1}{1}$	$2\frac{2}{1}$.	*13.*	$\frac{1}{1}$	M. 39.	*24.*	$\frac{1}{1}\frac{1}{1}$	F. 57.
3.	$\frac{1}{1}$	F. 6.	*14.*	$\frac{1}{1}$	M. 39.	*25.*	$\frac{1}{1}\frac{1}{1}$	M. 59.
4.	$\frac{1}{1}$	F. 28.	*15.*	$\frac{1}{1}\frac{1}{1}$	F. 43.	*26.*	$\frac{1}{1}\frac{1}{1}$	M. 59.
5.	$\frac{1}{1}$	M. 30.	*16.*	$\frac{1}{1}\frac{1}{1}$	M. 45.	*27.*	$\frac{1}{1}\frac{1}{1}$	M. 59.
6.	$\frac{1}{1}$	F. 30.	*17.*	$\frac{1}{1}\frac{1}{1}$	M. 45.	*28.*	$\frac{1}{1}\frac{1}{1}$	M. 61.
7.	$\frac{1}{1}$	M. 33.	*18.*	$\frac{1}{1}\frac{1}{1}$	M. 47.	*29.*	$\frac{1}{1}\frac{1}{1}$	F. 62.
8.	$\frac{1}{1}$	F. 33.	*19.*	$\frac{1}{1}\frac{1}{1}$	M. 49.	*30.*	$\frac{1}{1}\frac{1}{1}$	M. 67.
9.	$\frac{1}{1}$	F. 36.	*20.*	$\frac{1}{1}\frac{1}{1}$	F. 50.	*31.*	$\frac{1}{1}\frac{1}{1}$	M. 68.
10.	$\frac{1}{1}$	F. 36.	*21.*	$\frac{1}{1}\frac{1}{1}$	M. 51.	*32.*	$\frac{1}{1}\frac{1}{1}$	F. 89.
11.	$\frac{1}{1}$	M. 38.	*22.*	$\frac{1}{1}\frac{1}{1}$	F. 54.			

Males	18
Females	13
Sex unknown	1

It can be seen that this list includes all the sets in which the left lateral sinus is the larger, but leaves six over:

1. $\frac{1}{1}$ F. 6, where the right lateral sinus is somewhat the larger and the mastoid viens are equal.

2. $\frac{1}{1}$ F. 30, where the bones do not allow of comparison of the lateral sinuses and mastoid veins.

On the left side only in—

$\frac{1}{1}\frac{1}{1}\frac{1}{1}$ F. 62.

On both sides in—

$\frac{1}{1}\frac{1}{1}$ M. 33.

The size of the veins varies enormously; one side may be larger than the other, the larger being frequently associated with the larger lateral sinus, as in—

$\frac{1}{1}$ F. 37, where the right lateral sinus is the larger, the left mastoid vein being rudimentary,

but not always so, for the right lateral sinus may be the larger with the smaller mastoid vein, as in—

$\frac{1}{1}\frac{1}{1}$ M. 28,

$\frac{1}{1}\frac{1}{1}$ M. 56.

The vein may be indistinguishable on both sides, as in—

$\frac{1}{1}\frac{1}{1}$ M. 47,

$\frac{1}{1}\frac{1}{1}$ F. 89;

or it may be very small on both sides, as in—

$\frac{1}{1}\frac{1}{1}$ M. 48,

or indistinguishable on one side and very small in the other, as in—

$\frac{1}{1}\frac{1}{1}$ F. 54, the left being very small and the right indistinguishable.

It may be double at the start and exit on both sides, as in—

$\frac{1}{1}\frac{1}{1}$ F. 76.

It may be double at the start and exit on one side and single at the start and exit on the other, as in—

$\frac{1}{1}$ F. 22, the right side being double.

It may be single at the start and double at the exit on both sides as in—

$\frac{1}{1}\frac{1}{1}$ M. 33.

It may be single at the start and double at the exit on one side and single at the start and exit on the other, as in—

$\frac{1}{1}$ F. 36, where the left is double at the exit.

$\frac{1}{1}$ M. 37, where the left is double at the exit.

$\frac{1}{1}\frac{1}{1}$ F. 43, where the right is double at the exit.

$\frac{1}{1}\frac{1}{1}$ M. 82, where the right is double at the exit.

It may be double at the start and single at the exit on one side and single at both start and exit on the other, as in—

3. $\frac{84}{87}$ M. 39, where the left sulcus is somewhat the larger; both lateral sinuses are large, the right being slightly the larger, but the right mastoid vein coming off above the knee of the sinus s larger than the left, which is rudimentary.

4. $\frac{105}{106}$ F. 43, where the right mastoid vein and lateral sinus are the larger, but the left mastoid vein is rudimentary.

5. $\frac{113}{114}$ M. 45, in which set the bones do not allow of comparison of the lateral sinuses and mastoid veins.

6. $\frac{127}{128}$ M. 49, where both sulci are small, but the left is decidedly larger than the right. The right lateral sinus is the larger, but has a large mastoid vein, while the left mastoid vein is rudimentary.

Nos. 3, 4 and 6 of the above suggest that the size of the sulcus may sometimes depend on the size of the mastoid vein. Other sets also support this:

$\frac{33}{36}$ M. 28,

$\frac{108}{116}$ M. 45,

in both of which sets the right lateral sinus and sulcus are the larger, the left sulcus being out of all proportion smaller than the right, and the left mastoid vein being much larger.

$\frac{219}{220}$ M. 71, where both sulci are large and high lying and both mastoid veins are small.

But against such a suggestion are the following sets, which also show that a large lateral sinus does not always mean a large sulcus:

$\frac{183}{184}$ M. 60, where the right lateral sinus is large and the mastoid vein and sulcus are small, and—

$\frac{203}{204}$ M. 64, where both lateral sinuses are large and both sulci small, the right mastoid vein being rudimentary and the left small.

The position of the sulcus influences the spread of cells inwards as in—

$\frac{63}{64}$ M. 35,
$\frac{103}{104}$ M. 43,
$\frac{139}{140}$ M. 51,

where the smaller sulcus allows of a larger spread of cells under the labyrinth.

High out-pushing sulci are seen in all types of bone.

(*b*) *Marked bulging of cells forming a bulla internally to, and behind, the mastoid process* (*digastric bulla*) is seen in thirteen sets:

1. $\frac{25}{26}$ F. 21 on both sides, more marked on the right
2. $\frac{29}{30}$ M. 24 on the right side only.
3. $\frac{39}{40}$ F. 28 on both sides.
4. $\frac{103}{104}$ M. 43 on the right side only.
5. $\frac{131}{132}$ F. 49 on both sides.
6. $\frac{139}{140}$ M. 51 on both sides, more marked on the left.
7. $\frac{141}{142}$ M. 51 on both sides.
8. $\frac{145}{146}$ M. 52 on the right side only.
9. $\frac{195}{196}$ F. 62 on both sides, more marked on the left, where it runs up to the sulcus jugularis.
10. $\frac{207}{208}$ F. 64 on both sides, more marked on the left.
11. $\frac{209}{210}$ M. 66 on both sides.
12. $\frac{235}{236}$ M. 82 on the left side only.
13. $\frac{237}{238}$ F. 86 on both sides, more marked on the right.

In all these pus is liable to perforate into the neck internally to the mastoid process.

(*c*) *Specially large idgastric fossæ* are seen in four sets:

1. $\frac{41}{42}$ F. 28, the left being the larger.
2. $\frac{49}{50}$ M. 30 on both sides.
3. $\frac{53}{54}$ M. 33 on the right side, the left not allowing of examination.
4. $\frac{59}{60}$ F. 33 on the right side, the left not allowing of examination.

2. THE INTERIOR

(*i*) *Types*

Classification of the types is founded on the conditions of the outer antral wall and mastoid process. In referring to the mastoid process the terms "upper and lower mastoid"

are used, the "upper mastoid" being the bone below the level of the apex of the antrum down to the upper level of the projecting part or "lower mastoid."

This division is justified by the totally different surgical anatomical relationships.

On this basis it is found that of the 120 sets 82 are symmetrical and 38 asymmetrical. In many of the asymmetrical bones there is a tendency to symmetry.

It may be here stated that there are very few cellular bones without remains of diploë, either as a small mass at the extreme tip of the mastoid process, or as a rim round the tip.

Asymmetry as to extension of cells is indicated with each specimen.

Symmetrical

(*a*) Dense outer antral wall lined internally with cells which can be seen at the eighth month of fœtal life, and therefore called fœtal cells, with a dense layer of bone between the antrum and a diploëtic mastoid. *Diploetic infantile type*.....24

For my description of this type and its surgical importance reference can be made to the Transactions of the Eighth International Congress of Otology held in Buda-Pesth; in that paper, the importance of the type in conducing to intra-cranial complications in acute infection of the antrum and to chronic middle-ear suppuration, and its sequelæ, apart from tuberculosis, was pointed out.

1.	$\frac{1}{2}$	M.	$\frac{5}{12}$.	*9.*	$\frac{41}{12}$	F.	28.	*17.* $\frac{148}{16}$ F. 53.
2.	$\frac{3}{4}$	F.	$1\frac{7}{12}$.	*10.*	$\frac{45}{12}$	M.	30.	*18.* $\frac{143}{14}$ F. 54.
3.	$\frac{5}{8}$	?	$1\frac{5}{12}$.	*11.*	$\frac{71}{12}$	F.	36.	*19.* $\frac{155}{16}$ F. 55.
4.	$\frac{7}{8}$	M.	2.	*12.*	$\frac{84}{12}$	M.	39.	*20.* $\frac{148}{16}$ M. 58.
5.	$\frac{11}{12}$	?	$2\frac{3}{12}$.	*13.*	$\frac{101}{12}$	F.	42.	*21.* $\frac{141}{14}$ M. 60.
6.	$\frac{13}{12}$	M.	$4\frac{5}{12}$.	*14.*	$\frac{107}{12}$	M.	44.	*22.* $\frac{213}{14}$ M. 67.
7.	$\frac{14}{12}$	F.	6.	*15.*	$\frac{118}{12}$	M.	45.	*23.* $\frac{217}{16}$ M. 68.
8.	$\frac{11}{11}$	M.	28.	*16.*	$\frac{117}{11}$	F.	50.	*24.* $\frac{231}{11}$ F. 77.

Males	12
Females	10
Sex unknown	2

Children up to 6 years of age..... 7
Adults.....17

(*b*) Dense outer antral wall and very few cells in the upper mastoid. Diploëtic lower mastoid..... 1

1. $\frac{11}{11}$ M. 24.

(*c*) Dense outer antral wall. Cellular upper mastoid. Diploëtic lower mastoid..... 5

1. $\frac{11}{11}$ M. 20.
2. $\frac{17}{17}$ F. 22.
3. $\frac{18}{18}$ M. 30.
4. $\frac{111}{112}$ M. 56.
5. $\frac{101}{101}$ M. 64.

(*d*) Dense outer antral wall. Upper and lower mastoid cellular..... 1

$\frac{101}{102}$ F. 61. More diploë at the tip of the left mastoid than on the right.

(*e*) Diploëtic outer antral wall. A few cells in upper mastoid. Lower mastoid diploëtic..... 1

$\frac{203}{204}$ M. 64.

(*f*) Cellular outer antral wall and upper mastoid. Lower mastoid diploëtic.....12

1. $\frac{9}{10}$ F. 2.
2. $\frac{17}{18}$ M. 30.
3. $\frac{64}{66}$ F. 35.
4. $\frac{99}{100}$ M. 42.
5. $\frac{118}{120}$ M. 48.
6. $\frac{143}{144}$ M. 51.
7. $\frac{147}{148}$ M. 53.
8. $\frac{165}{166}$ M. 57.
9. $\frac{171}{172}$ M. 58.
10. $\frac{183}{184}$ M. 60.
11. $\frac{221}{222}$ M. 72.
12. $\frac{223}{224}$ M. 72.

(*g*) Cellular outer antral wall and entire mastoid, some having more dipoë at the tip than the others, and some more one side than the other.....38

1. $\frac{21}{21}$ F. 16.
2. $\frac{25}{26}$ F. 21. Digastric bulla on both sides, more marked on the right.
3. $\frac{23}{24}$ M. 24. Digastric bulla on the right side only.
4. $\frac{33}{34}$ F. 25.
5. $\frac{39}{40}$ F. 28. Digastric bulla on both sides.
6. $\frac{51}{52}$ F. 30.
7. $\frac{61}{62}$ M. 34.

8. $\frac{63}{64}$ M. 35. The occipital diploë is invaded by cells on the right side and not on the left.

9. $\frac{67}{68}$ M. 36.

10. $\frac{69}{70}$ F. 36.

11. $\frac{79}{80}$ M. 38.

12. $\frac{81}{82}$ M. 38. Extension inwards, behind the middle ear under the labyrinth and invading the lower part of the internal diploë on both sides.

13. $\frac{83}{84}$ F. 38.

14. $\frac{85}{86}$ F. 38.

15. $\frac{89}{90}$ M. 39. The cells on the left side only extend inwards behind the middle ear and under the labyrinth.

16. $\frac{91}{92}$ M. 39.

17. $\frac{103}{104}$ M. 43. The cells extending inwards under the labyrinth are larger on the left side where the sulcus jugularis is not so high.
Digastric bulla on the right side only

18. $\frac{117}{118}$ M. 48.

19. $\frac{123}{124}$ F. 48. Mastoid cortex dense and thick on the right side and thin on the left.

20. $\frac{131}{132}$ F. 49. Digastric bulla on both sides.

21. $\frac{133}{134}$ M. 50.

22. $\frac{135}{136}$ F. 50.

23. $\frac{139}{140}$ M. 51. The cells extend over the meatus into the zygoma on both sides. Extension inwards behind the middle ear under the labyrinth and invading the lower part of the internal diploë on both sides, but more on the right, on which side the sulcus jugularis is not so high. Digastric bulla on both sides, more marked on the left.

24. $\frac{141}{142}$ M. 51. Digastric bulla on both sides, more marked on the right.

25. $\frac{159}{160}$ F. 55.

26. $\frac{167}{168}$ F. 57.
27. $\frac{178}{180}$ M. 59.
28. $\frac{182}{186}$ M. 61.
29. $\frac{188}{190}$ F. 61.
30. $\frac{195}{196}$ F. 62. Digastric bulla on both sides, more marked on the left, where it runs inwards to the sulcus jugularis.
31. $\frac{197}{198}$ F. 62.
32. $\frac{207}{208}$ F. 64. Digastric bulla on both sides, more marked on the left.
33. $\frac{208}{210}$ M. 66. Digastric bulla on both sides.
34. $\frac{211}{212}$ F. 66. Cells extending up into the squama on the right side only.
35. $\frac{212}{216}$ M. 71.
36. $\frac{224}{226}$ F. 72.
37. $\frac{235}{236}$ M. 82. Digastric bulla on the left side only.
38. $\frac{237}{238}$ F. 86. Digastric bulla on both sides, more marked on the right.

Males 18
Females 20
Total 38
Total symmetrical 82

Asymmetrical

In many of the asymmetrical sets it will be seen that there is a decided tendency to symmetry.

(*a*) Diploëtic infantile type on one side (*a*).
Fine cells in the outer antral wall and diploëtic mastoid on the other; a rare condition (*b*) 2

1. (*a*) $\frac{13}{14}$ F. 29.
2. (*b*) $\frac{198}{200}$ F. 63.

In these sets suppuration is likely to perforate the outer antral wall on the left side.

(*b*) Diploëtic infantile type on one side (*a*). Dense outer antral wall with a few cells in the upper part of a diploëtic mastoid on the other (*b*) 5

1. $\frac{(b)10}{(a)26}$ M. 11.
2. $\frac{(b)47}{(a)56}$ M. 33.

3. $\frac{(a)77}{(b)78}$ F. 37.
4. $\frac{(a)127}{(b)128}$ M. 49.
5. $\frac{(b)187}{(a)183}$ M. 61.

(*c*) Diploëtic infantile type, on one side (*a*). Dense outer antral wall, cellular upper mastoid and diploëtic lower mastoid, on the other (*b*).....3

1. $\frac{(a)35}{(b)36}$ M. 28.
2. $\frac{(b)115}{(a)116}$ M. 47.
3. $\frac{(a)121}{(b)122}$ F. 48.

(*d*) Diploëtic infantile type, on one side (*a*). Cellular outer antral wall and upper mastoid and diploëtic lower mastoid, on the other (*b*).....5

1. $\frac{(a)93}{(b)94}$ M. 39.
2. $\frac{(b)129}{(a)130}$ M. 49.
3. $\frac{(a)151}{(b)152}$ F. 54.
4. $\frac{(b)173}{(a)174}$ M. 59.
5. $\frac{(a)233}{(b)234}$ F. 81.

(*e*) Diploëtic infantile type, on one side (*a*). Cellular outer antral wall and entire mastoid, on the other (*b*).....5

1. $\frac{(a)97}{(b)98}$ F. 41.
2. $\frac{(b)105}{(a)106}$ F. 43. The cells in the right are small and surrounded by dense bone; diploë at the tip of the mastoid.
3. $\frac{(b)163}{(a)164}$ F. 56.
4. $\frac{(a)193}{(b)194}$ F. 61.
5. $\frac{(a)227}{(b)228}$ F. 73. Densely diploëtic on the right.

(*c*), (*d*), and (*e*) explain why, in acute bilateral antral infection, a mastoid abscess may result on one side and a chronic discharge without a mastoid abscess on the other.

Those specimens which have only a dense outer wall and a very few cells in the upper part of a diploëtic mastoid have the same surgical importance with regard to the suppuration as the pure diploëtic type, and the same applies to those bones in which the entire mastoid is composed of cells surrounded by a thick layer of dense bone.

There are, therefore, twenty sets in which the diploëtic infantile type is seen on one side only. On the right side in twelve, and on the left side in eight.

Nine are in males and eleven in females.

It is interesting to note that in the whole series of a hundred and twenty individuals the type is seen in forty-four; both sides in twenty-four, and on one side in twenty.

(*f*) Dense outer antral wall, cellular upper mastoid, and diploëtic lower mastoid, on one side (*a*).

Dense outer antral wall and entirely cellular mastoid, on the other (*b*).....3

1. $\frac{(a)113}{(b)114}$ M. 45.
2. $\frac{(a)206}{(b)208}$ F. 64.
3. $\frac{(a)238}{(b)240}$ F. 89.

(*g*) Dense outer antral wall, cellular upper mastoid and diploëtic lower mastoid, on one side (*a*).

Cellular outer antral wall and upper mastoid and diploëtic lower mastoid, on the other (*b*).....3

1. $\frac{(a)73}{(b)74}$ M. 37. Very few cells in the upper mastoid on the right side.
2. $\frac{(a)111}{(b)112}$ M. 45. Very few cells in the upper mastoid on the right side
3. $\frac{(b)156}{(a)157}$ F. 55.

(*h*) Dense outer antral wall, cellular upper mastoid and diploëtic lower mastoid, on one side (*a*).

Cellular outer antral wall and entire mastoid on the other. (*b*).....2

1. $\frac{(a)125}{(b)126}$ M. 49.
2. $\frac{(a)175}{(b)176}$ M. 59.

(*i*) Dense outer antral wall and entire mastoid cellular, on one side (*a*).

Cellular outer antral wall and entire mastoid, on the other (*b*).....1

1. $\frac{(b)87}{(a)88}$ M. 39.

(*j*) Cellular outer antral wall and upper mastoid, lower mastoid diploëtic, on one side (*a*).

Cellular outer antral wall and entire mastoid, on other (*b*).....6

1. $\frac{(a)17}{(b)18}$ M. 9.
2. $\frac{(b)53}{(a)54}$ M. 33.
3. $\frac{(b)145}{(a)146}$ M. 52. On the right side, the cells extend inwards from the mastoid, forming a digastric bulla.

4. $\frac{(b)}{(a)}\frac{177}{177}$ M. 59.
5. $\frac{(b)}{(a)}\frac{111}{111}$ M. 68.
6. $\frac{(b)}{(a)}\frac{111}{111}$ F. 76.

These sets show that in double mastoid infection the lower mastoid is not necessarily affected on both sides.

Diploë in the outer antral wall is very unusual; it is present on one side only in the three following sets. Reference has already been made to $\frac{111}{111}$ M. 64, where diploë is present in the outer antral wall on both sides.

(*k*) Diploëtic outer antral wall and entire mastoid, on side (*a*).

Dense outer antral wall with a few cells just below the apex of the antrum, the remainder of the mastoid being diploëtic, on the other (*b*).....2

1. $\frac{(b)}{(a)}\frac{11}{11}$ M. 33.

The left bone in this set is a rare and interesting one. The section shows the diploë in the zygomatic squamous and petrous elements, separated from one another by distinct partitions of compact bone, and the squamous diploë forming the outer antral wall, which is half an inch in thickness, is separated from the cells which always line the outer antral wall (fœtal cells) by another thin layer of compact bone. On the right side the dense outer antral wall is formed by thickening of the compact bony partition between the zygomatic and squamous masses of diploë.

This type of bone is responsible for those cases of infection from the antrum which run an osteomyelitic course.

2. $\frac{(a)}{(b)}\frac{22}{22}$ F. 33.

The right bone of this set is very like the left one of the previous set, but the layer of dipoë in the outer antral wall is much thinner and the compact layer between the diploë and the fœtal cells is much thicker.

(*l*) *3.* Diploëtic outer antral wall, cellular upper mastoid and diploëtic lower mastoid, on one side (*a*)

Dense outer antral wall with cells in the base of the upper mastoid, the remainder of which is diploëtic, on the other (*b*).....1

1. $\frac{(b)7\frac{1}{2}}{(a)7\frac{6}{6}}$ M. 37.

The left bone shows a thin outer layer of diploë formed by the zygomatic squamous and mastoid elements with distinct thin separating layers of compact bone. The zygomatic element forms the outer antral wall, the antrum being high lying.

Total asymmetrical.....38

(ii) *The size and shape of the antrum* are subject to very slight variation on the two sides; a difference, and that a small one, is only seen in two sets—

1. $\frac{1\,2\,7}{1\,0\,6}$ M. 44,

2. $\frac{1\,4\,8}{1\,3\,6}$ F. 53,

In both of which sets the left antrum is smaller than the right, both bones being of the diploëtic infantile type.

(iii) *High lying antrum* in relation to the posterior zygomatic line is seen in nineteen sets:

1. $\frac{3\,7}{3\,6}$ M. 28. Bilateral diploëtic infantile type.

2. $\frac{3\,2}{4\,0}$ F. 28. Higher on the right side.

3. $\frac{3\,3}{3\,4}$ M. 33.

4. $\frac{4\,1}{4\,2}$ M. 34.

5. $\frac{7\,3}{7\,4}$ M. 37. Higher on the right side, where the bone is nearly of the pure diploëtic infantile type for there is only one small cell in the mastoid diploë.

6. $\frac{7\,5}{7\,6}$ M. 37.

7. $\frac{8\,7}{8\,8}$ M. 39.

8. $\frac{9\,1}{9\,2}$ M. 39.

9. $\frac{1\,0\,3}{1\,0\,4}$ M. 43.

10. $\frac{1\,1\,1}{1\,1\,2}$ M. 45.

11. $\frac{1\,2\,8}{1\,4\,0}$ M. 51.

12. $\frac{1\,4\,7}{1\,4\,8}$ M. 53. On the right side only.

13. $\frac{1\,7\,1}{1\,7\,6}$ M. 59. Higher on the right side.

14. $\frac{1\,8\,3}{1\,8\,4}$ M. 60.

15. $\frac{1\,8\,5}{1\,8\,6}$ M. 61. On the right side only.

16. $\frac{1\,9\,5}{1\,9\,6}$ F. 62. Higher on the right side.

17. $\frac{2\,1\,3}{2\,1\,4}$ M. 67. Bilateral diploëtic infantile type.

18. $\frac{2\,2\,1}{2\,2\,2}$ M. 72.

19. $\frac{2\,3\,3}{2\,3\,4}$ M. 72.

It is in the great majority of cases symmetrical, for in two sets only is the antrum high lying on one side and not on the other:

$\frac{1}{1}\frac{1}{1}\frac{1}{1}$ M. 53,

$\frac{1}{1}\frac{1}{1}\frac{1}{1}$ M. 61,

Where the right in each instance is the high lying one.

In four sets one antrum is higher than the other, and the higher one is, in all, on the right side:

1. $\frac{1}{1}\frac{1}{1}$ F. 28.

2. $\frac{1}{1}\frac{1}{1}$ M. 37.

3. $\frac{1}{1}\frac{1}{1}$ M. 59

4. $\frac{1}{1}\frac{1}{1}$ F. 62.

It is more common in males than in females, for seventeen are males and only two are females.

It is most commonly seen in the cellular bones, for in two sets only is it present in the diploëtic infantile type, and in them on both sides

1. $\frac{1}{1}\frac{1}{1}$ M. 28.

2. $\frac{1}{1}\frac{1}{1}$ M. 67.

The antrum may be high lying and yet the middle fossa may dip down, partly shutting off the cavity from the surface as, on the right side, in

$\frac{1}{1}\frac{1}{1}$ M. 45.

There is no reliable external guide to a high lying antrum, except that a posterior zygomatic line which slopes downwards and backwards should put one on one's guard.

(iv) *The sinus tympani* or internal pyramidal recess cannot be examined in all specimens, but the sets,

$\frac{1}{1}\frac{1}{1}$ M. 58,

$\frac{1}{1}\frac{1}{1}$ M. 64,

deserve careful study, for an extraordinarily large cavity is seen on both sides in each. This cavity may be infected in suppuration and responsible for chronic discharge with or without antral infection and for the continuation of discharge after a radical operation has been performed. Unless the cavities are as large as these, it is practically impossible to deal with them without injury to the facial nerve; but if they are large they can be opened immediately below the external semi-circular canal and behind the level of the descending

part of the facial nerve, cleaned of contents and laid well open into the bony wound, as in a case of a boy, aged 9 years, described by myself in the 'Transactions of the Royal Society of Medicine, Otological Section,' vol. iv, No. 4, p. 54.

It is interesting to note that Mr. G. J. Jenkins considers this cavity as the upper end of the first branchial cleft.

The diagnosis of disease in this cavity cannot be dealt with in this paper.

(v) *Malformation of the bony external semi-circular canal* is seen in 191, the right bone of a woman, aged 61 years. The canal is represented by a single large cavity opening widely into the outer wall of the vestibule. A small tubercle on the upper part of the sloping posterior wall of the cavity represents the only attempt at the formation of the normal partition.

The canals on the left side are normal.

B. In Disease

(i) *Injuries*

Fracture through the bone is seen in three sets only.

1. *23.* The right bone of a male, aged 20 years. The fracture passes through the middle ear and posterior part of the labyrinth dividing the facial nerve.
2. *180.* The left bone of a male, aged 59 years. The fracture passes through the middle ear, meatus, and mastoid cells just externally to the external semi-circular canal. The facial nerve is not divided.
3. *184.* The left bone of a male, age 60 years The fracture passes right across the bone just externally to the external semi-circular canal dividing the facial nerve in its descending course.

(ii) *Changes in bone*

(*a*) *Exostoses in the meatus* on both sides more marked on the right in—

$\frac{184}{156}$ M. 57.

(*b*) ***A pedunculated exostosis*** is present on the inner wall of a large cell *in the lower mastoid* of the right bone of—

179. M. 59.

(*c*) ***Hyperostosis of the tympanic plate*** on both sides, in—

$\frac{21\,8}{11\,6}$ M. 68.

(*d*) ***Thinning of the tympanic plate***, due to the presence of masses of epithelium, keratosis obturans, is seen on both sides of—

$\frac{4\,4}{6\,6}$ F 38,

being more marked on the left. Evidence of past suppuration is seen in both membranes.

(*e*) ***Bony excrescences on the inner aspect of the bone*** are seen in five sets, all associated with mental trouble:

1 $\frac{4\,2}{5\,0}$ F 30. A general paralytic. An exostosis is seen on the lower edge of the lateral sinus groove on the right side only.

2. $\frac{4\,4}{6\,6}$ F. 35. An epileptic dement. The excrescences are present on the inner surface of the squama on both sides, but more marked on the left. There is also great thickening of the parietal bone.

3. $\frac{7\,5}{7\,6}$ M. 37. An epileptic dement. An esostosis is present on the lower edge of the lateral sinus groove, on the left side only. The markings of the cerebral convolutions are very pronounced on both sides.

4. $\frac{1\,3\,1}{1\,3\,2}$ F 49. A melancholic. The excrescences are seen on the inner aspect of the squama, on the left side especially.

5. $\frac{3\,3\,7}{3\,3\,6}$ F 86 A senile dement. The same as the preceding specimen.

(*f*) ***Great thickening of the skull with corrugations and deep groovings*** due to osteitis deformans, is seen on both sides in—

1. $\frac{3\,3\,3}{3\,3\,0}$ F. 76.

The thickening is seen in the parietal bones, the temporal bone escaping altogether, but the inner aspect of the squama is corrugated and deeply grooved for vessels.

(*g*) ***Extreme dilatation, with thinning of the left internal***

***auditory meatus*, due to the presence of a cerebellar tumour, is seen in 52, the left bone of a female, aged 30.**

(*h*) ***Fixation of the stapes*** is seen in—

$\frac{11}{12}$ F. 36.

This patient had been getting deaf since childhood. For many years, about eight, she had been insane with subacute mania. Death was due to pulmonary and intestinal tuberculosis. She had very marked hallucinations of hearing, hearing voices and chattering to imaginary persons. She also gave a rambling and confused account of a medical student whom she loved and who had promised to marry her after he had operated on her for deafness. She spoke in a slow wailing monotone.

Her father had been deaf since boyhood and at one time heard better in a noise.

Both bones are of the diploëtic infantile type, and both membranes are intact.

Right bone.—Outer vestibular wall.

(*i*) From the vestibular side. The posterior half of the base of the stapes appears thickened and pushed well through the oval window and fixed, while the anterior is not thickened and appears a little tilted out from the window as if the stapedius muscle had exerted a permanent effect. The anterior half of the upper lip of the oval window is much thickened.

(*ii*) From the tympanic side. The inner half of the crura of the stapes are buried in new tissue, the bone being absolutely fixed.

Left bone.—Outer vestibular wall.

(*i*) From the vestibular side. Very similar to the right side.

(*ii*) From the tympanic side. The crura of the stapes (fractured accidentally) are not so buried as on the right side, but the base is firmly fixed.

It is interesting to note that both bones are of the diploëtic infantile type.

(*iii*) ***Evidence of suppuration is seen in thirteen sets.*** The type of bone on both sides in each set is described.

(*a*) Evidence in the membrane without sign of antral infection.

1. $\frac{7}{11}$ M. 37. *Right bone.*—Calcareous deposit in the anterior and posterior segments. No sign of antral infection. Dense outer antral wall with a few cells in the upper part of a diploëtic mastoid.

   Left bone.—Crescentic calcareous deposit, with a dry perforation in the anterior inferior segment.

   Ossicles intact. No sign of antral infection. Diploëtic and cellular outer antral wall, cellular upper mastoid, diploëtic lower mastoid.

   In the writer's opinion calcareous deposits are always the result of a pyogenic infection.

2. $\frac{11}{11}$ F. 38. *Right bone.*—Cicatrix in the posterior segment.

   Tympanic plate thinned by the presence in the meatus of layers of epithelium (keratosis obturans). No sign of antral infection. Cellular outer antral wall and mastoid, the tip of which is diploëtic.

   Left one.—Dry perforation in the posterior superior segment.

   Ossicles intact. Meatus markedly dilated, the tympanic plate being very much thinned by keratosis obturans. No sign of antral infection.

   Cellular outer antral wall and entire mastoid.

These two sets point to the fact that the antrum does not always share in infection of the lower middle ear, for if the cavity was ever infected in them, it is difficult to understand why an acute mastoid abscess did not result. The cavity is more likely to be primarily implicated in virulent infection due to scarlet fever, measles, and influenza. In specimens like these, however, a fresh or the old infection may at any time spread to the antrum and mastoid cells, producing acute mastoid signs and symptoms.

3. $\frac{181}{182}$ M. 30.—*Right bone,*—Membrane was intact, but injured on section. No sign of infection. Diploëtic infantile type.

Left bone.—Large dry inferior kidney shaped perforation. Ossicles intact. No sign of antral infection.

Diploëtic infantile type.

In this left bone it would have been impossible for a mastoid abscess to form if the antrum had been also infected.

(*b*) Evidence in the membrane only on one side and in the antrum as well on the other.

1. $\frac{87}{88}$ F. 41. *Right bone.*—Dry perforation in the inferior segment. Ossicles intact. No sign of antral infection.

Dense outer antral wall, entire mastoid cellular.

Left bone.—Anterior and posterior segments cicatricial.

No perforation. Ossicles intact. The antral walls are smoothed out as if the cavity had at one time been infected. No cholesteatoma. Diploëtic infantile type.

In this right bone, infection of the antrum must have led to mastoid disease, this again pointing to the fact that the antrum does not always share in infection with the lower middle ear. In the left, the signs of infection of the antrum seem to be clear and yet healing had occurred, and if mastoid cells had been present mastoid disease would have certainly resulted.

2. $\frac{118}{119}$ M. 48. *Right bone.*—Dry inferior perforation. Ossicles intact. No sign of antral infection. Cellular outer antral wall and mastoid, the tip of which is diploëtic.

Left bone.—Posterior inferior perforation. Ossicles intact.

The lining membrane of the antrum and mastoid cells appears thickened.

Cellular outer antral wall and mastoid, the cells being surrounded by dense bone.

In this right bone again if the antrum had shared in the infection of the lower middle ear mastoid disease must have resulted.

In the cellular left bone, chronic discharge had been present, but, as previously stated, a bone with mastoid cells surrounded by dense bone is as conducive to a chronic middle-ear discharge as is the pure diploëtic infantile type.

(*c*) Evidence in the middle ear and antrum on bothsides.

1. $\frac{3}{5}\frac{5}{6}$ M. 39. *Right bone.*—Pus in the middle ear. Perforation in Shrapnell's membrane. Loss of the incus and head of the malleus. Erosion of the outer attic wall. Stapes intact Cholesteatoma in the attic and filling the antrum. Smoothing out of the antral walls. Diploëtic infantile type.

Left bone.—Pathological conditions exactly similar to those on the right side, except that there are calcareous deposits in the anterior and inferior segments of the membrane.

Diploëtic infantile type.

This set demonstrates how symmetry of anatomical type produces symmetrical pathological conditions.

(*d*) Evidence in the middle ear and antrum on one side, the other side being normal.

1. $\frac{3}{3}\frac{1}{2}$ M. 24. *Right bone.*—Normal. Membrane intact. Dense outer antral wall; a few cells are present in the upper part of a diploëtic mastoid.

Left bone.—The radical operation has been performed and the middle fossa opened.

Death occurred from cerebellar abscess; the pathway of infection is unknown.

Diploëtic infantile type.

This left bone illustrates the fact that the diploëtic or infantile type is conducive not only to chronic suppuration, but to its more dangerous sequelæ.

2. $\frac{1}{1}\frac{8}{6}\frac{1}{2}$ F. 42. *Right bone.*—Normal. Membrane intact. Diploëtic infantile type.

Left bone.—Perforation in the posterior segment. Ossicles intact. No discharge. The inner and posterior antral walls are smoothed out.

Diploëtic infantile type.

It is interesting to note that healing had taken place without operation on the antrum.

3. $\frac{115}{115}$ M. 47. *Right bone.*—Normal. Membrane intact. Dense outer antral wall; cellular mastoid with a diploëtic tip.

Left bone.—Large loss of the membrana tensa. Loss of the articular process of the incus. Thickening of the antral lining membrane.

Diploëtic infantile type.

4. $\frac{137}{137}$ F. 50. *Right bone.*—Normal. Membrane intact.

Diploëtic infantile type.

Left bone.—Pus was present in the middle ear and antrum, which is small. Large loss of the membrana tensa.

Ossicles intact.

Diploëtic infantile type.

In this left bone, if simple treatment did not produce a cure, the trouble would probably have been amenable to an incomplete mastoid operation.

5. $\frac{153}{154}$ F. 54. *Right bone.*—Normal. Membrane was intact, but has split.

Diploëtic infantile type.

Left bone.—Pus in the middle ear and antrum. Perforation in Shrapnell's membrane and posterior segment. Loss of the head of the malleus, incus, and crura of the stapes. Erosion of the outer attic wall. Niche for the oval window filled by new tissue. All the walls of the antrum are smoothed out, the fœtal cells being completely destroyed.

There was no cholesteatoma.

Diploëtic infantile type.

6. $\frac{137}{138}$ M. 49. *Right bone.*—Pus in the middle ear and antrum.

Complete loss of the membrane, malleus, incus and crura of the stapes, the base of which is intact, as can be seen from the vestibular side. The oval window niche is filled with new tissue. Marked erosion of the outer attic and posterior superior deep meatal walls; the inner and posterior antral walls are smoothed out. The mass of fœtal cells has been converted into a mass of dense bone, narrowing the aditus and antral cavity; this localised osteo-sclerotic condition can be easily distinguished from the normally dense outer antral wall. There was no cholesteatoma.

Diploëtic infantile type.

Left bone.—Normal. Membrane intact. The outer antral wall is thick and dense and the mastoid diploëtic, with the exception of a few small cells running downwards from the apex of the antrum.

7. $\frac{231}{232}$ F. 77. *Right bone.*—Pus in the middle ear and antrum.

Perforation was present in Shrapnell's membrane, but details were destroyed accidently on section. Erosion of the outer attic wall. The aditus is much narrowed by thickening of the free border of the squama of the petro-squamosal junction. The antral walls are smoothed out, the fœtal cells being completely destroyed up to the normal dense outer wall. The roof of the antrum is much thickened by osteo-sclerosis.

There was no cholesteatoma.

Diploëtic infantile type.

Left bone.—Normal; membrane intact.

Diploëtic infantile type.

This is the oldest specimen of chronic middle-ear suppuration which I possess. The woman died of senile mania, broncho-pneumonia, and heart failure.

(*e*) The two last sets have been placed together, for the affected bones show that a certain amount of osteo-sclerosis may take place in the walls of the antrum as a result of chronic suppuration, but it can always be distinguished from the normal dense outer antral wall, so characteristic of the normal diploëtic infantile type.

All the bones affected with chronic suppuration with antral implication are of the diploëtic infantile type, with the exception of *120* already described in which there are small mastoid cells surrounded by dense bone, a condition having the same surgical significance as the pure diploëtic infantile type, and, as may be seen in $\frac{101}{102}$F. 42, $\frac{137}{138}$ F. 50, $\frac{153}{154}$ F. 54, $\frac{231}{232}$ F. 77, suppuration was present in the middle-ear tract, including the antrum, on one side only, the other side being normal, and yet the diploëtic infantile type is present on *both* sides, thus adding emphasis to the fact that the conditions present in that type, viz. a dense outer antral wall, lined internally by the fœtal cells and a dense layer of bone separating the antrum from the diploëtic mastoid process, are factors in producing chronic middle-ear discharge, and the density is not a result.

To those who hold that the dense outer antral wall is the result of chronic suppuration, it can be pointed that the condition is present in this series without any signs of suppuration, on both sides in 18, and on one side in 16.

It can be confidently expected that in the future X-ray photography will be of the greatest value in the diagnosis and treatment of suppurative disease.

PHOTOGRAPHS ILLUSTRATING ARTHUR CHEATLE'S PAPER

ON

THE REPORT OF AN EXAMINATION OF BOTH TEMPORAL BONES FROM A HUNDRED AND TWENTY INDIVIDUALS IN REFERENCE TO THE QUESTION OF SYMMETRY IN HEALTH AND DISEASE

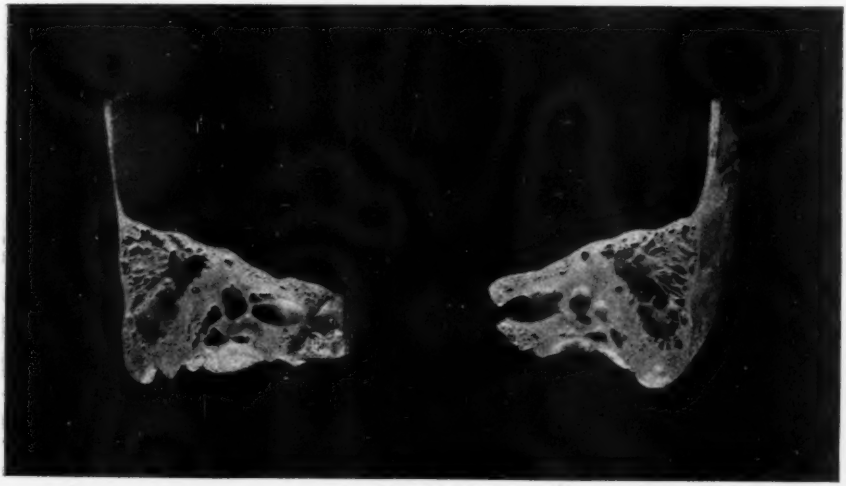
Presented at the Ninth International Otological Congress, Boston, 1912.

The Numbers of the Figures Have No Relation to Those Given in the Text.

No. 1.—FEMALE, AGED 2 YEARS.

Symmetrical.

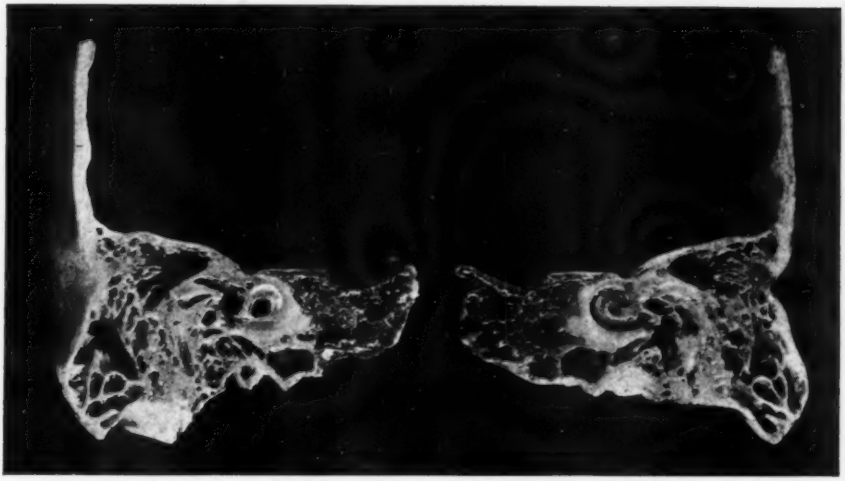
Right.—Long, narrow antrum. Diploe at the tip of the mastoid. Cells from the floor of the middle ear run under the first turn of the cochlea.



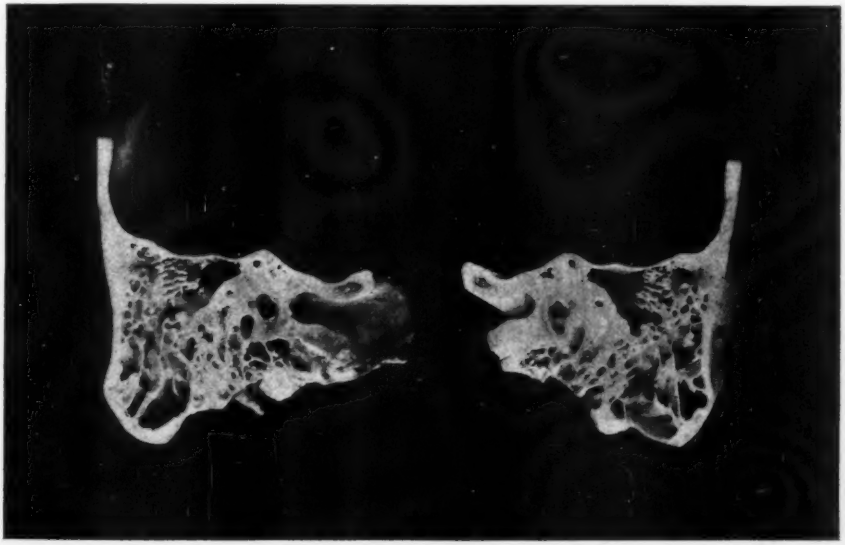
Left.—Symmetrical with the right. The section has been made further back than on the right side.

No. 2.—FEMALE, AGED 16 YEARS.

Symmetrical.



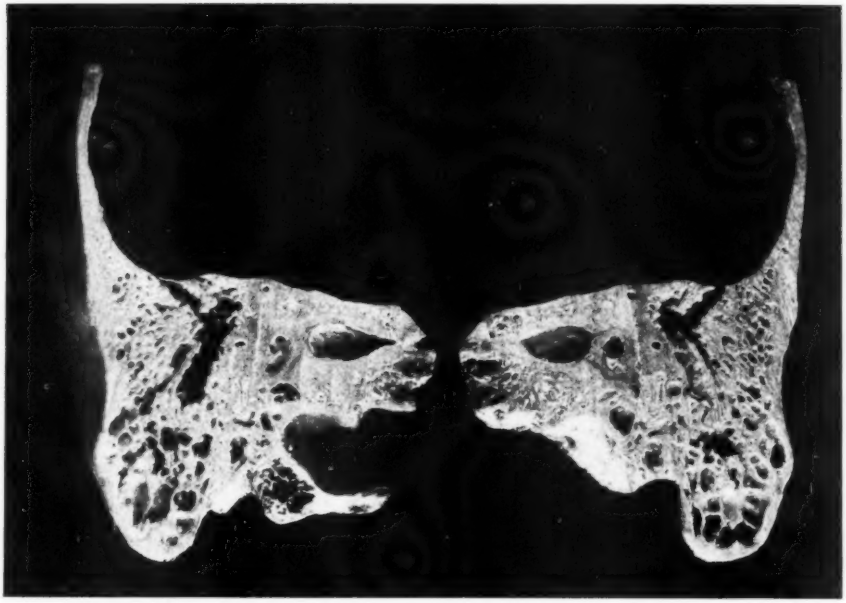
Right.—Outer antral wall and entire mastoid cellular. Cells from the floor of the middle ear pass under the cochlea, reaching and slightly invading the internal diploetic mass.



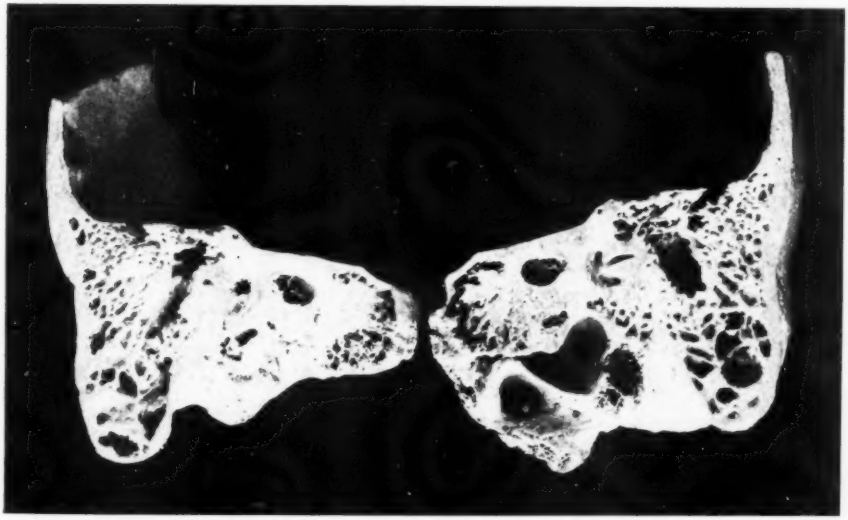
Left.—Symmetrical with the right.

No. 3.—FEMALE, AGED 49 YEARS.

Symmetrical.



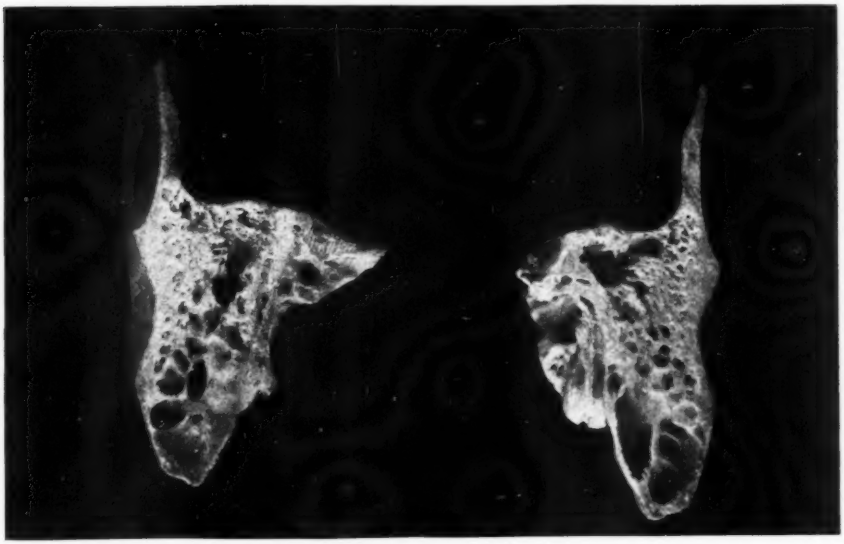
Right.—Outer antral wall and entire mastoid cellular. Long, narrow antrum.



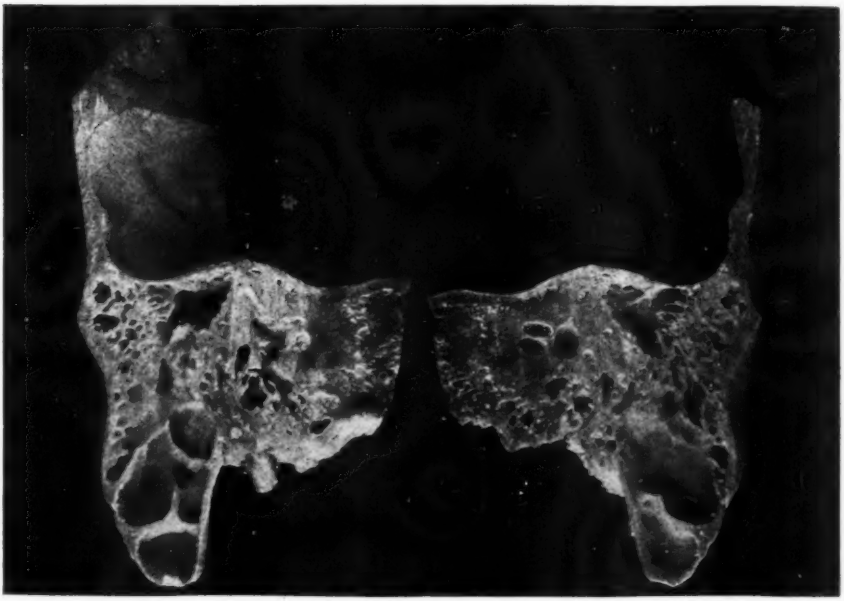
Left.—Symmetrical with the right.

No. 4.—MALE, AGED 50 YEARS.

Symmetrical.



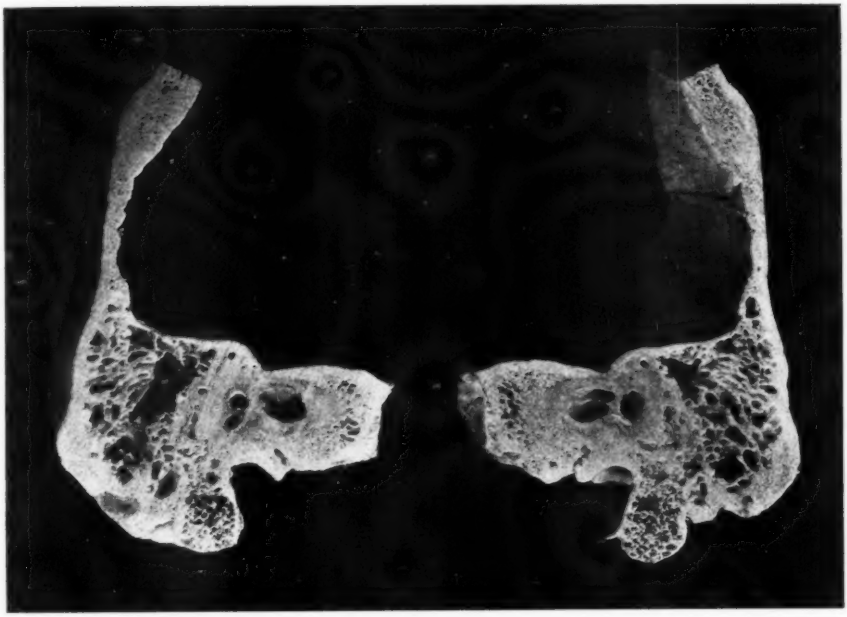
Right.—Outer antral wall and entire mastoid cellular. The lower cells are very large.



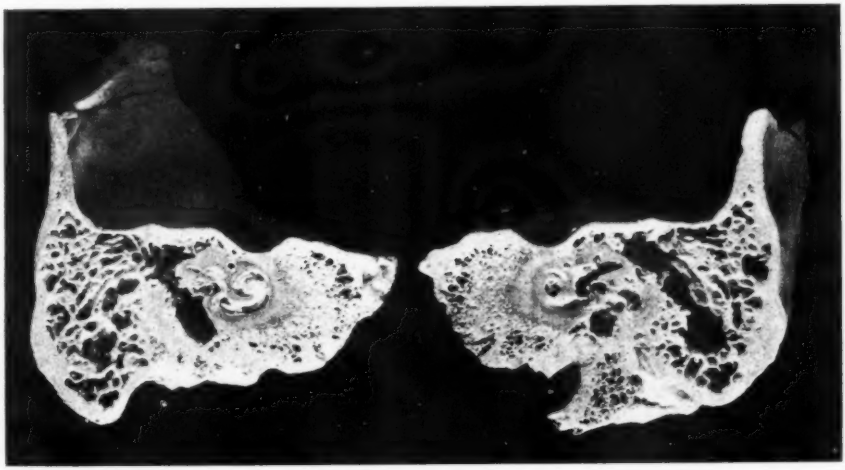
Left.—Symmetrical with the right.

No. 5.—FEMALE, AGED 55 YEARS.

Symmetrical.



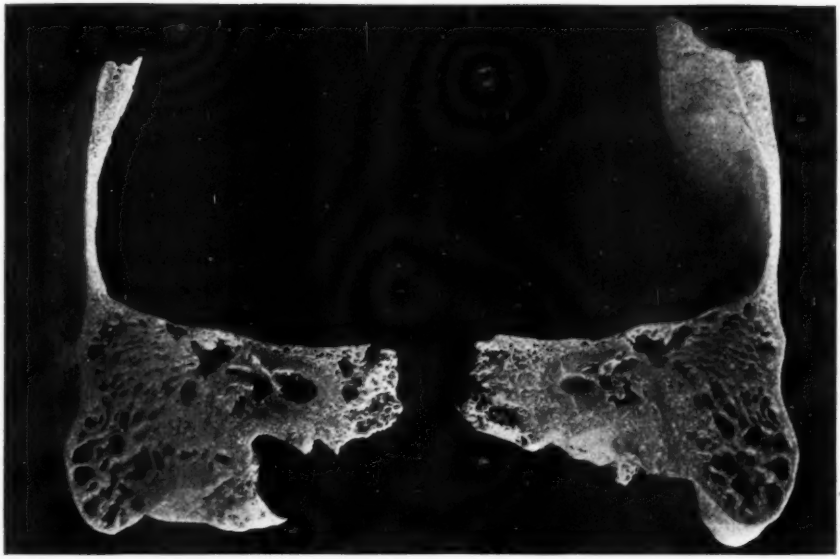
Right.—Outer antral wall and entire mastoid cellular. Long, narrow antrum. The cortex at the tip of the mastoid is very dense.



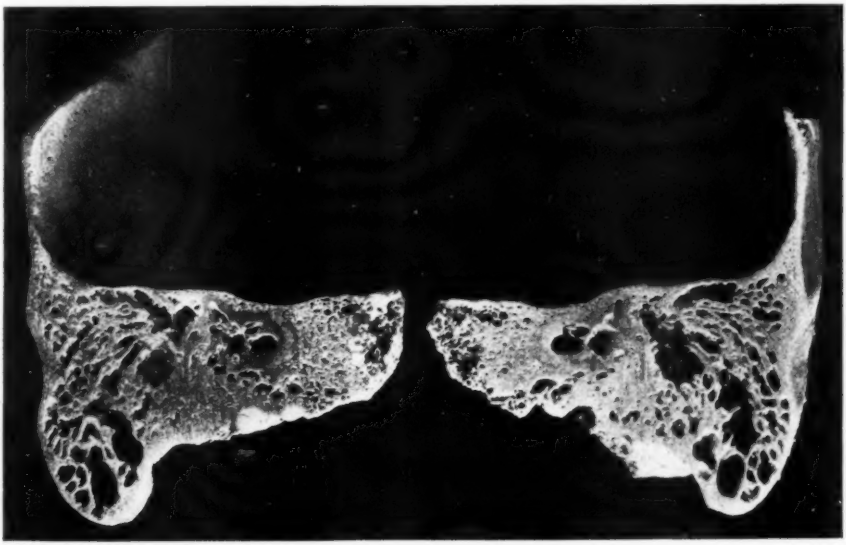
Left.—Symmetrical with the right. The cortex at the tip of the mastoid is not so dense as on the right side.

No. 6.—FEMALE, AGED 62 YEARS.

Symmetrical.



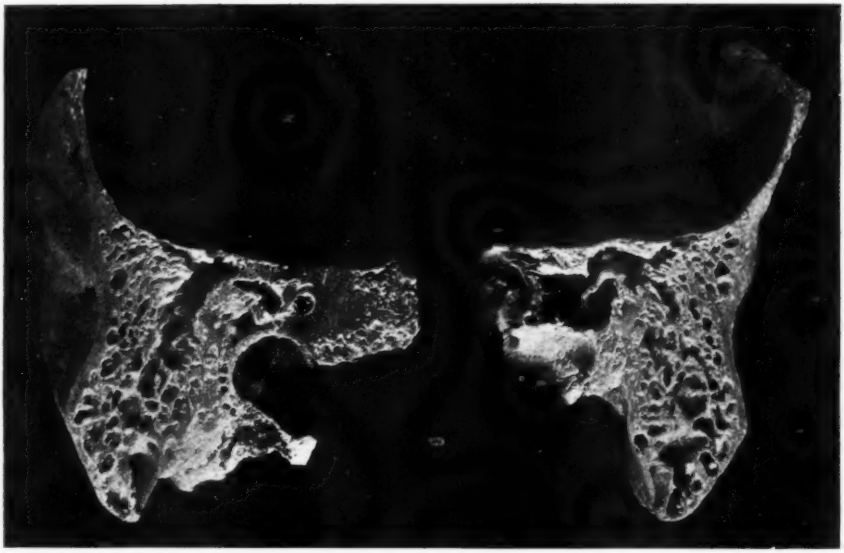
Right.—Outer antral wall and entire mastoid cellular. The antrum is slightly high lying.



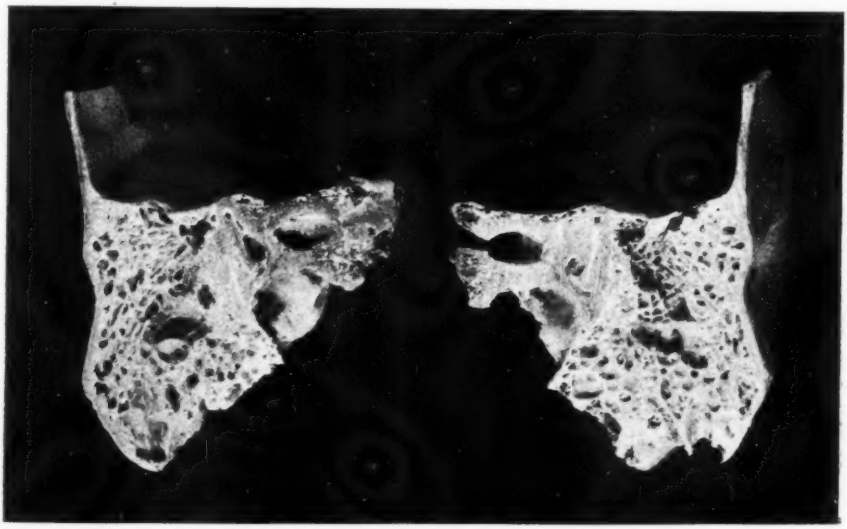
Left.—Symmetrical with the right.

No. 7.—MALE, AGED 41 YEARS.

Symmetrical.



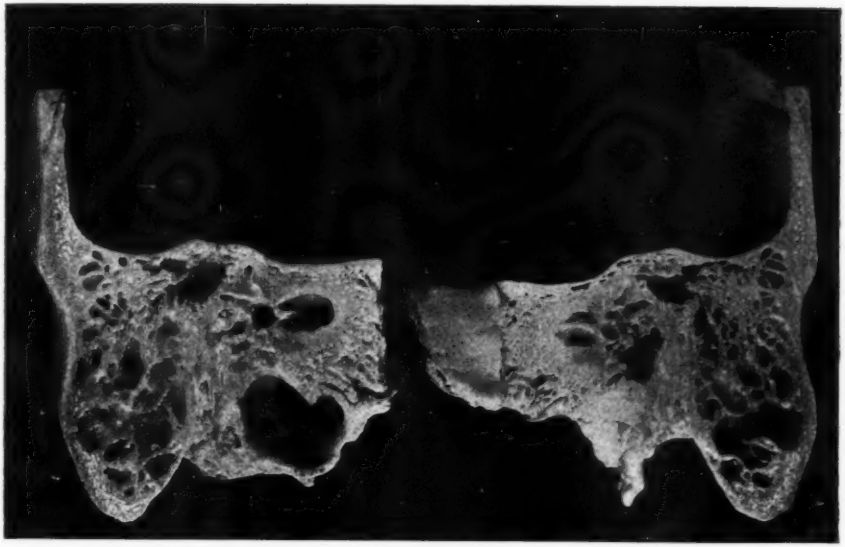
Right.—Outer antral wall and entire mastoid cellular. Long, narrow antrum.



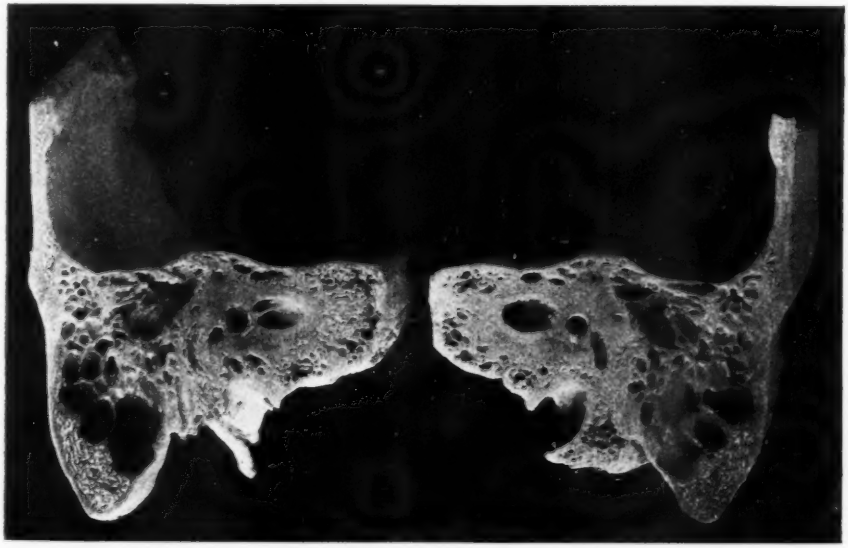
Left.—Symmetrical with the right.

No. 8.—MALE, AGED 36 YEARS.

Slight asymmetry.



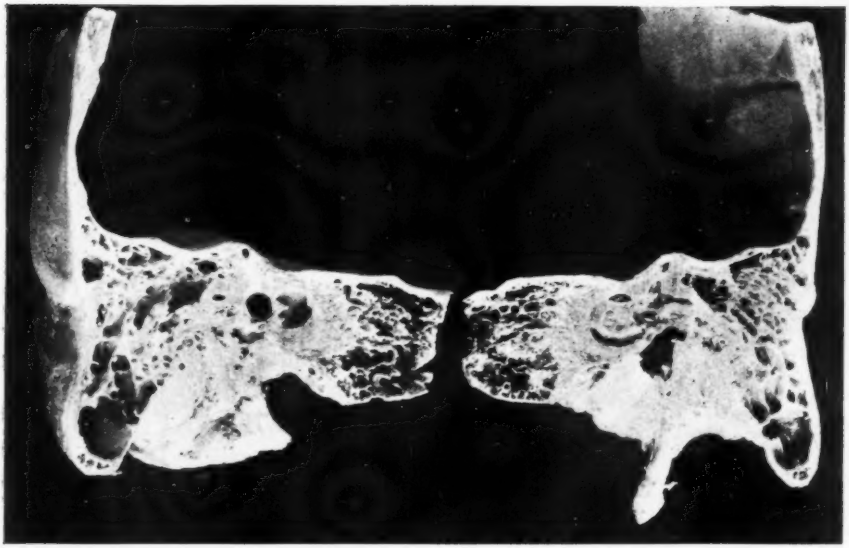
Right.—Outer antral wall and mastoid cellular, with a slight rim of diploe at the tip.



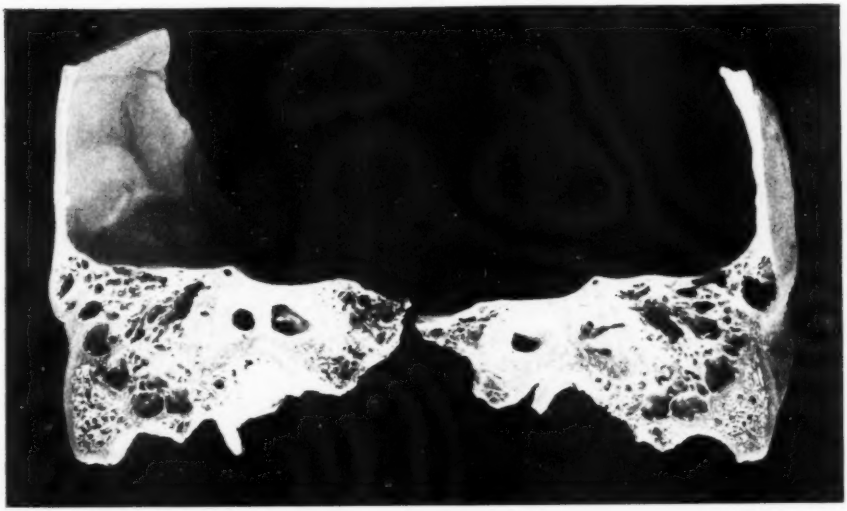
Left.—The same as the right, but there is more diploe at the tip of the mastoid.

No. 9.—MALE, AGED 33 YEARS.

Asymmetrical.

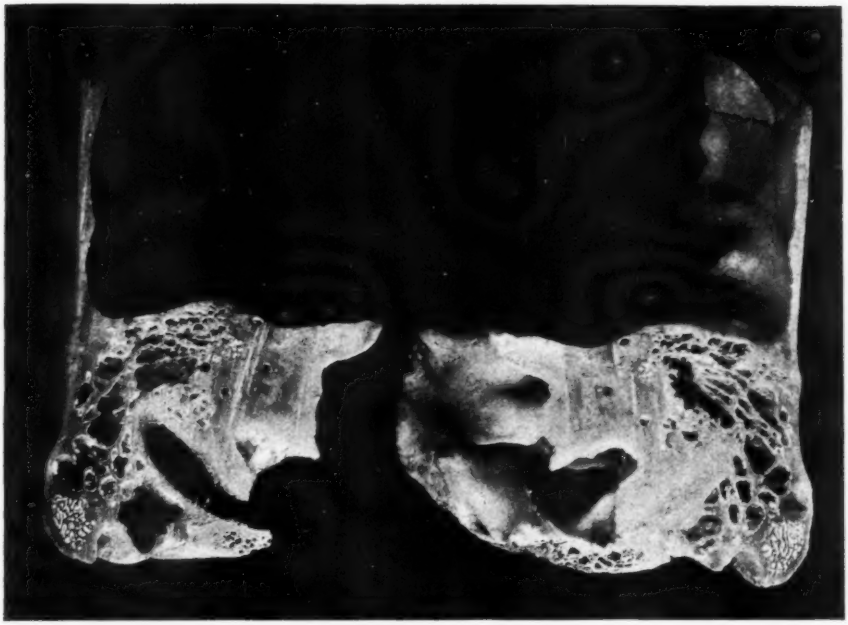


Right.—Outer antral wall and mastoid cellular, with a slight rim of diploe at the tip. Somewhat high-lying antrum.

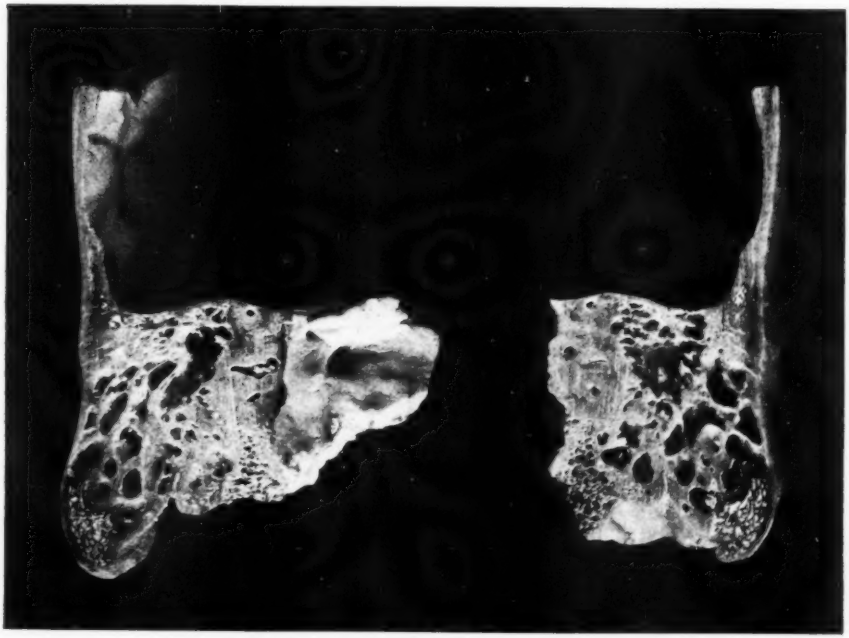


Left.—Outer antral wall and upper mastoid cellular. Lower mastoid is diploetic. There is a specially large cell in the zygomatic element. The cells run downwards and inwards over the digastric fossa. A thick layer of compact bone separates the cells from the diploe. Somewhat high-lying antrum.

Asymmetry of digastric bulla.



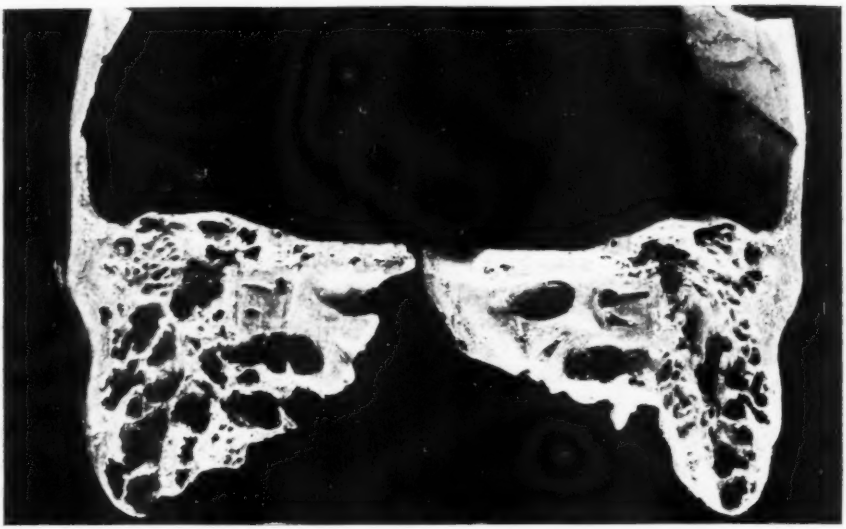
Right.—Outer antral wall partly dense and partly cellular. Upper mastoid cellular. Lower mastoid diploetic. Large digastric bulla.



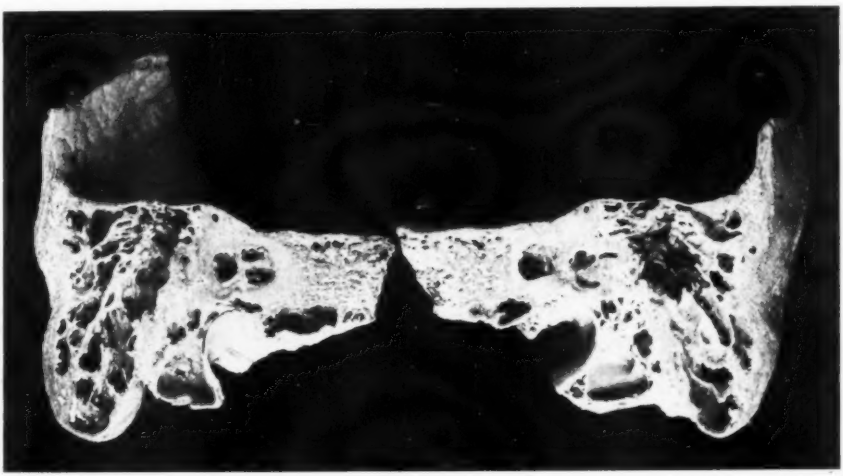
Left.—Symmetrical with the right, except that a smaller digastric bulla is present.

No. 11.—FEMALE, AGED 62 YEARS.

Occipital bulla on the left side only.



Right.—Cellular throughout. A small, low-lying sulcus jugularis allows the spread of cells inwards under the labyrinth.

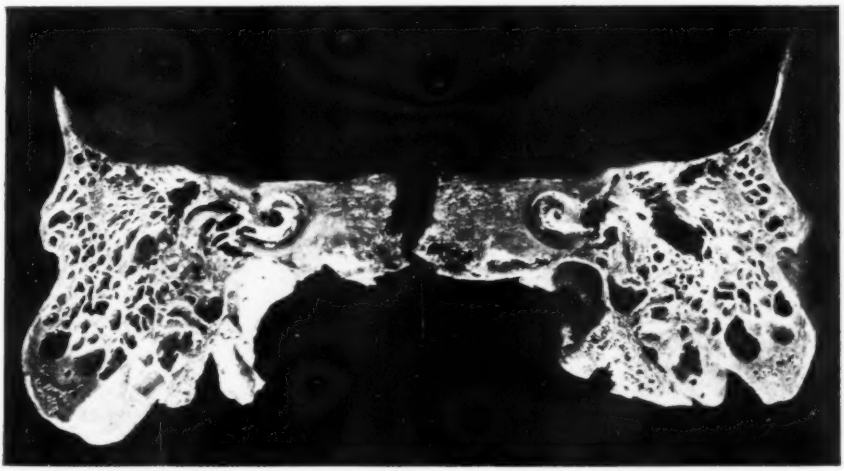


Left.—Cellular throughout. The sulcus jugularis is larger and lies higher than the right, only allowing a thin spread of cells inwards under the labyrinth. Cells invade the occipital, where a larger occipital bulla is present, lying against the outer wall of the sulcus jugularis.

No. 12.—MALE, AGED 51 YEARS.

Left sulcus jugularis the larger.

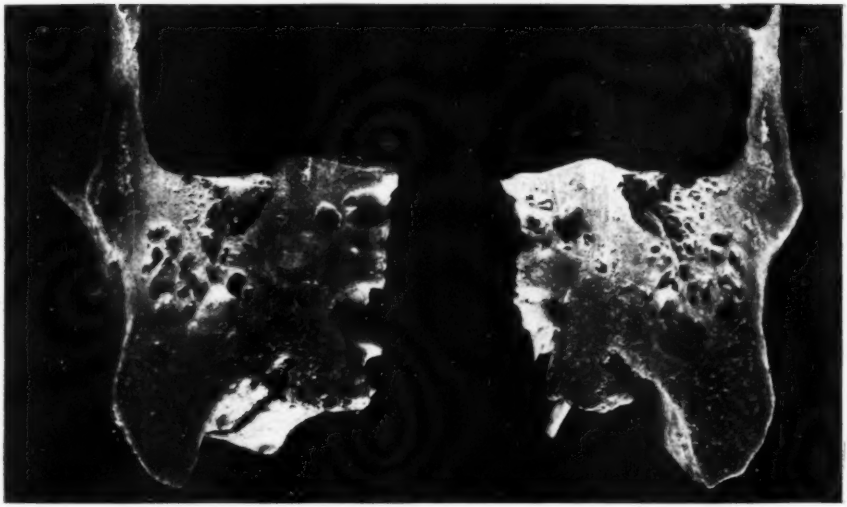
Right.—Cellular throughout. Long, narrow antrum. The cells extend inwards behind the middle ear, over a small, low-lying sulcus jugularis, and under the semicircular, vestibular and internal auditory meatus, and invade the lower part of the internal diploetic mass. The antrum is high lying to the posterior zygomatic line.



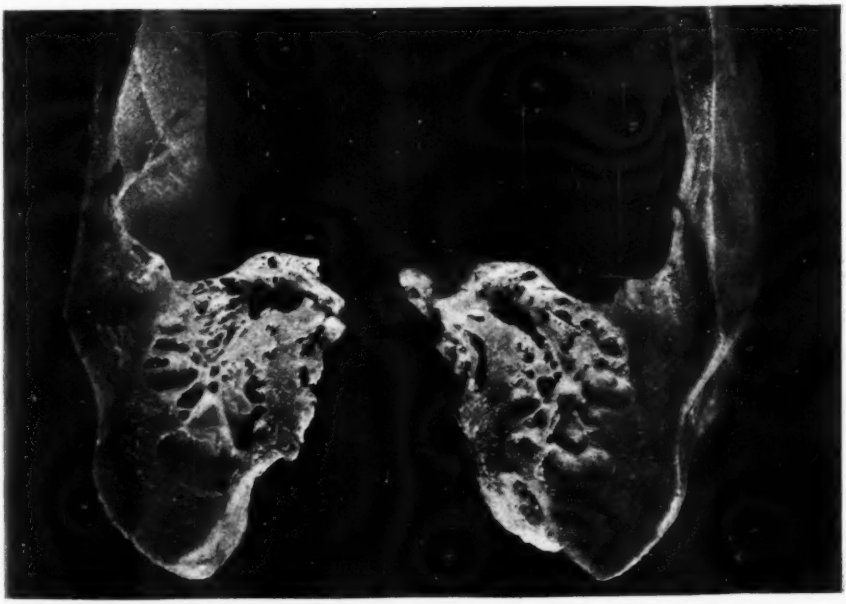
Left.—The antrum is not as long as on the right side, and diploe is present in the tip of the mastoid. The sulcus jugularis is larger and lies higher then on the right, lessening the cellular spread inwards. The cells run up to its outer wall. A digastric bulla is present. The antrum is high-lying.

No. 13.—MALE, AGED 50 YEARS.

Dipping down of the middle fossa on one side only, the left.



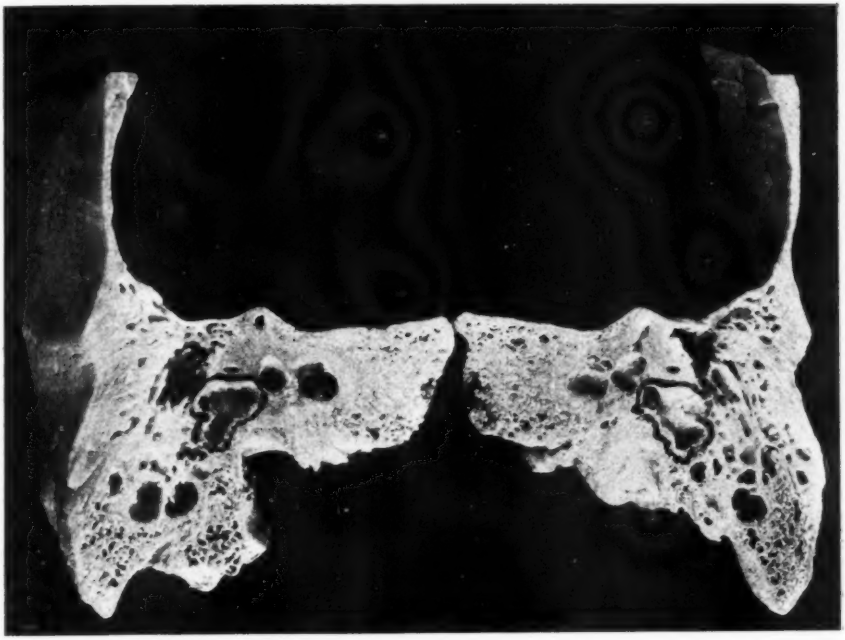
Right.—Outer antral wall dense. Upper mastoid cellular. Lower mastoid diploetic.



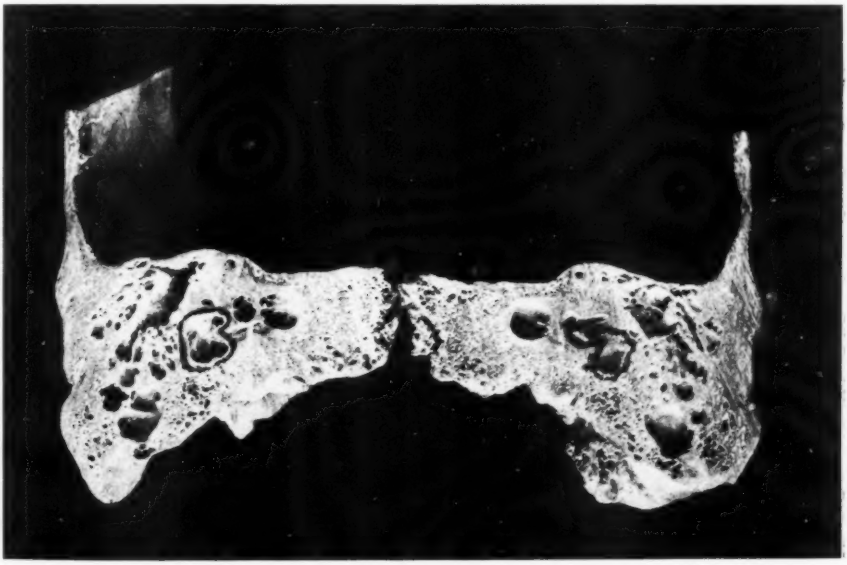
Left.—Interior symmetrical with the right, but the middle fossa dips down, causing a sloping roof to the antrum, a condition which is not present on the right side.

No. 14.—MALE, AGED 58 YEARS.

Symmetrical.



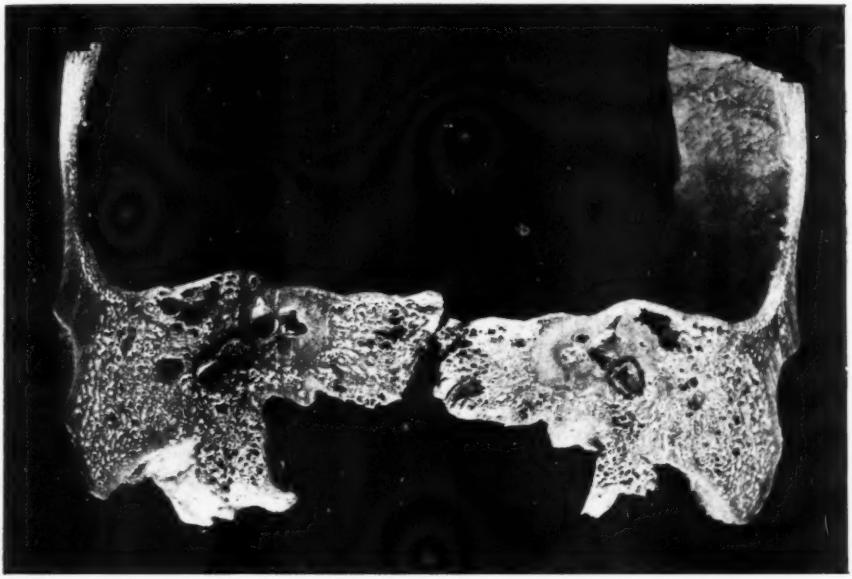
Right.—Showing an extensive sinus tympani (outlined in ink) with a small opening into the posterior middle ear wall.



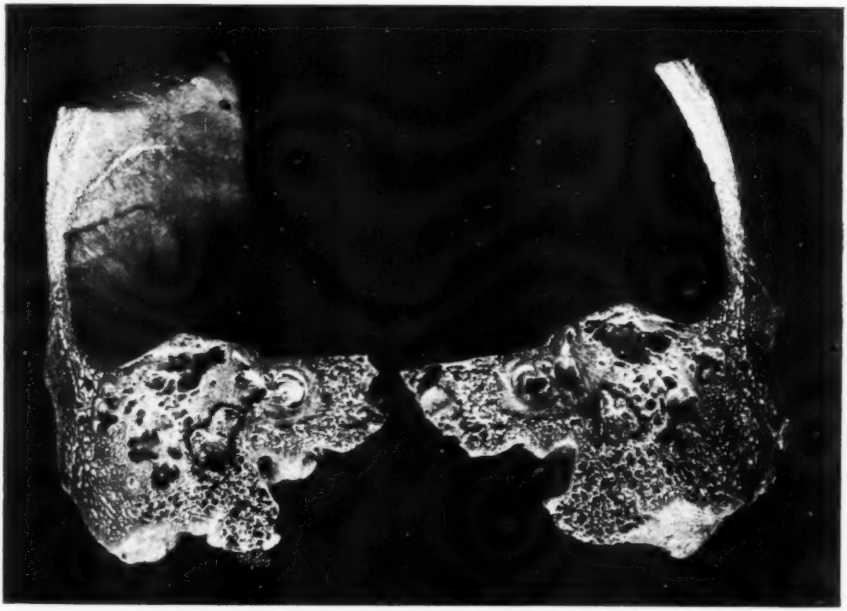
Left.—Symmetrical with the right.

No. 15.—MALE, AGED 64 YEARS.

Symmetrical.

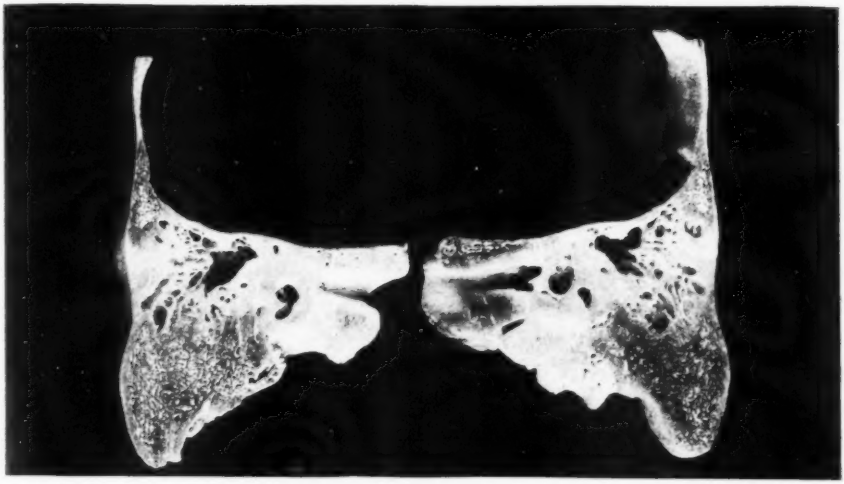


Right.—Showing a large sinus tympani, outlined in ink.

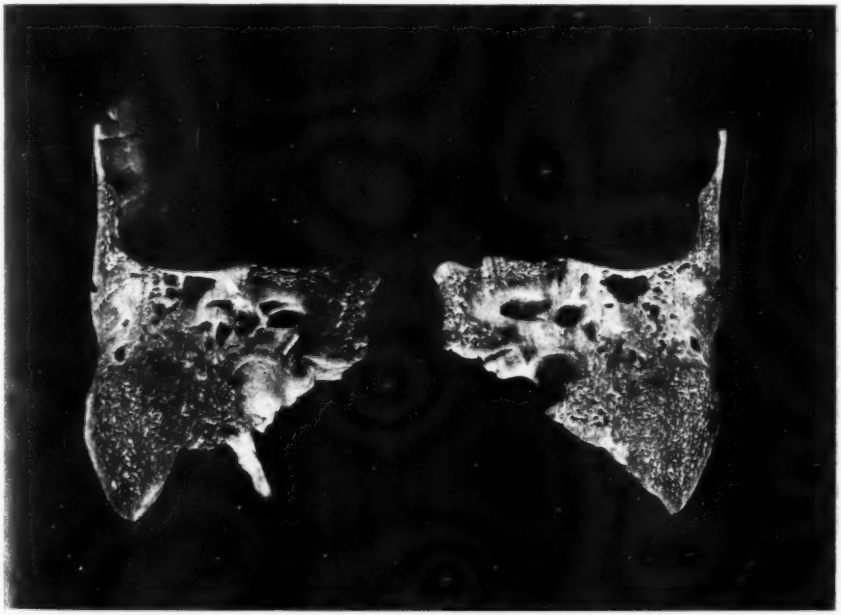


Left.—Symmetrical sinus tympani.

No. 16.—FEMALE, AGED 55 YEARS.



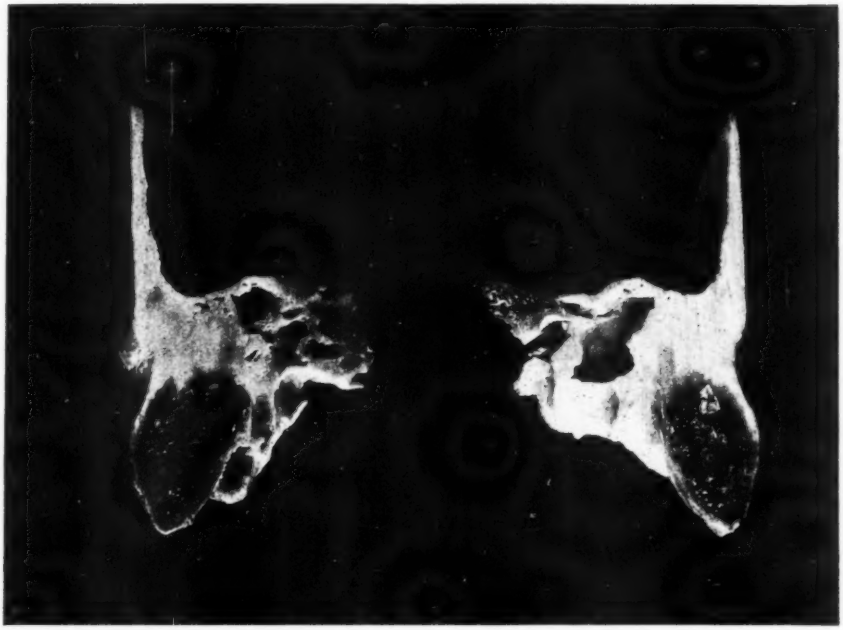
Right.—A few cells are present in the dense outer antral wall and in the upper part of the diploetic mastoid.



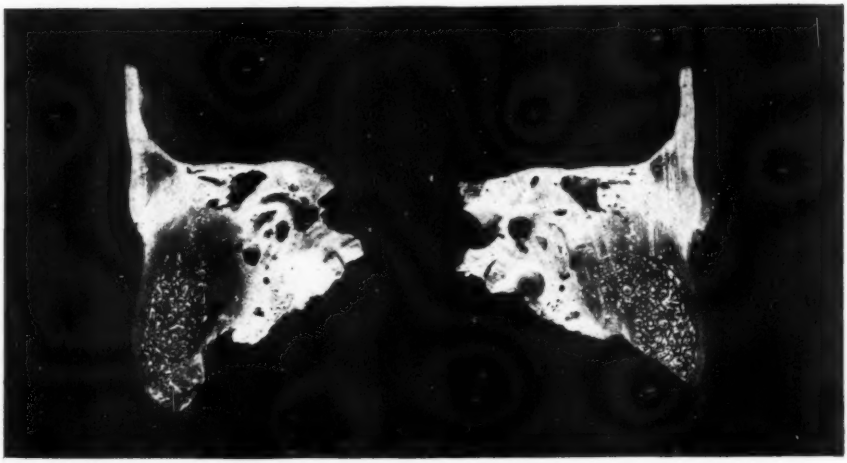
Left.—Symmetrical with the right, but there are fewer cells.

No. 17.—MALE, AGED 28 YEARS.

Symmetrical diploetic infantile type.



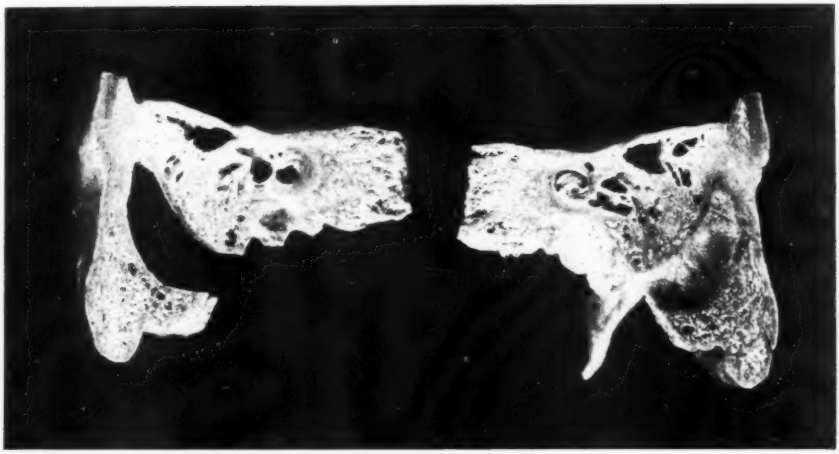
Right.—Diploetic infantile type.



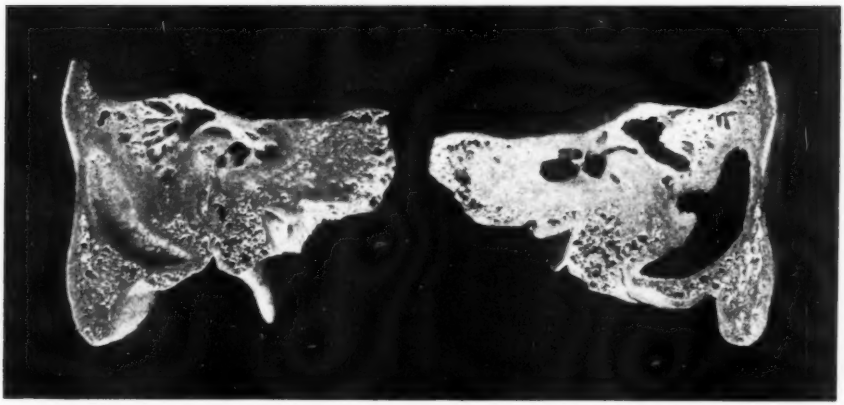
Left.—Diploetic infantile type, symmetrical with right.

No. 18.—MALE, AGED 45 YEARS.

Symmetrical diploetic infantile type.



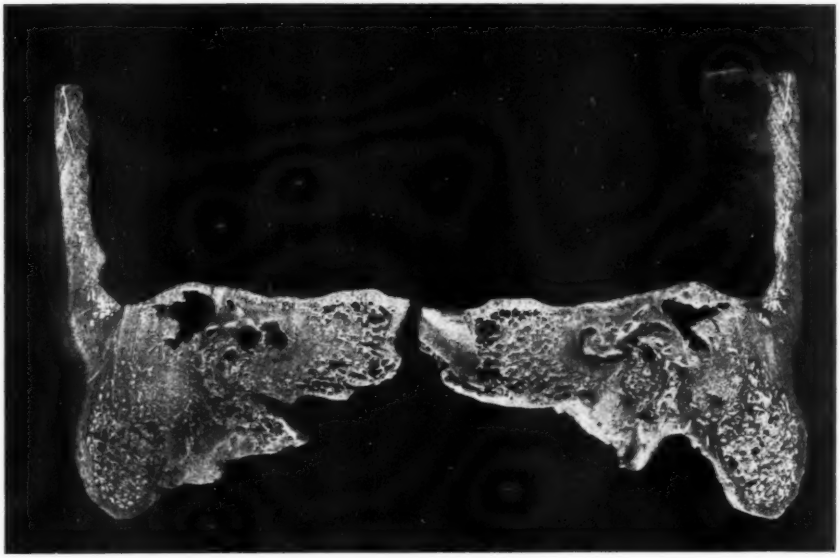
Right.—With very forward lateral sinus.



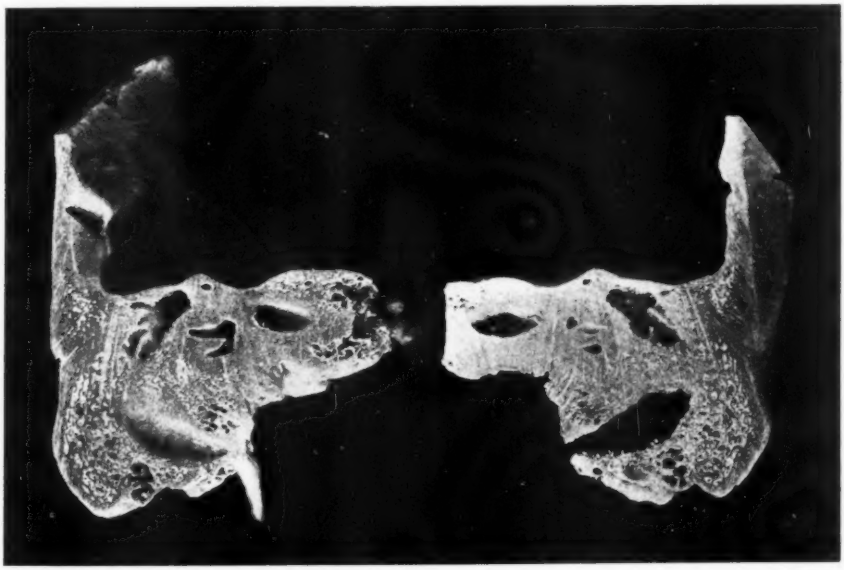
Left.—With very forward lateral sinus.

No. 19.—MALE, AGED 58 YEARS.

Symmetrical diploetic infantile type.



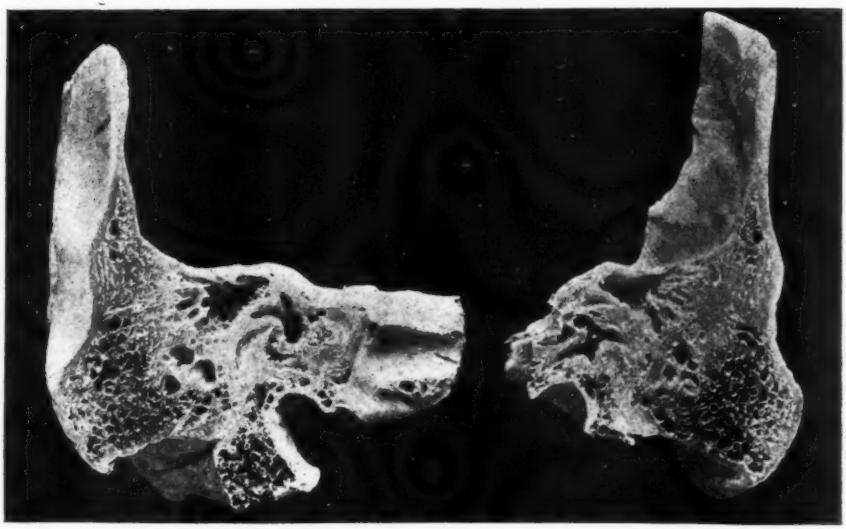
Right.—Bilateral diploetic type with dense outer antral wall and a dense layer between the antrum and diploe of the mastoid.



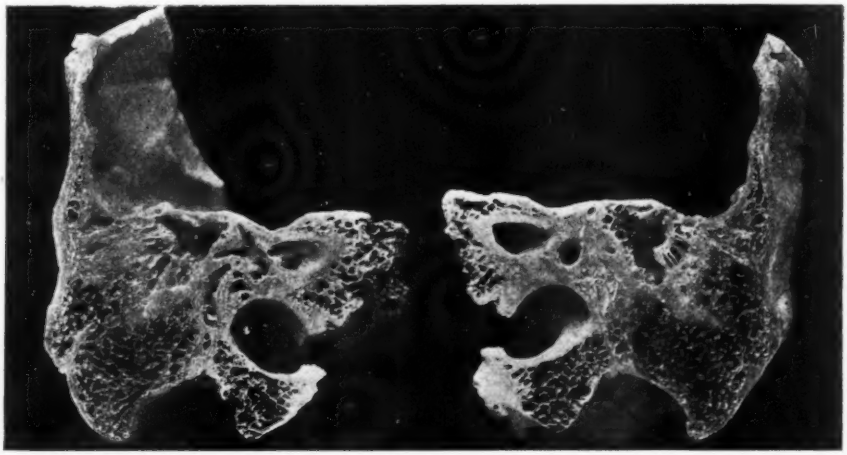
Left.—Symmetrical with right side.

No. 20.—MALE, AGED 33 YEARS.

Congenital imbecile.

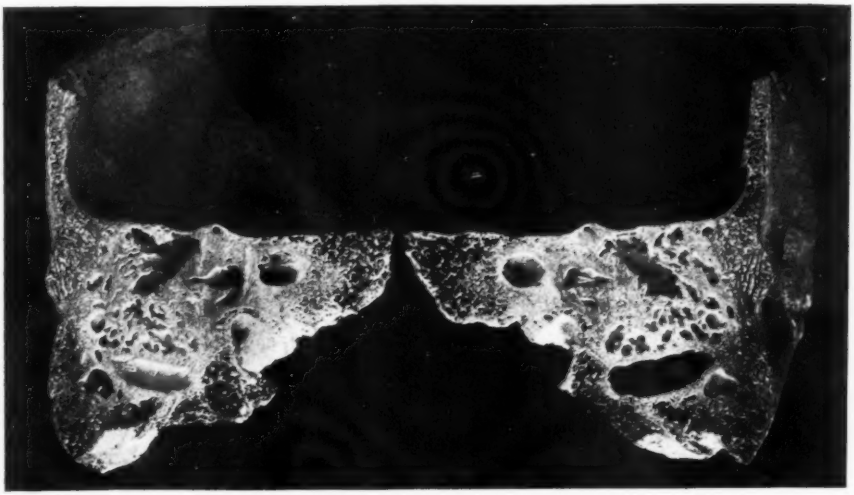


Right.—Outer antral wall dense. A few cells at the upper and inner aspect of a diploetic mastoid.

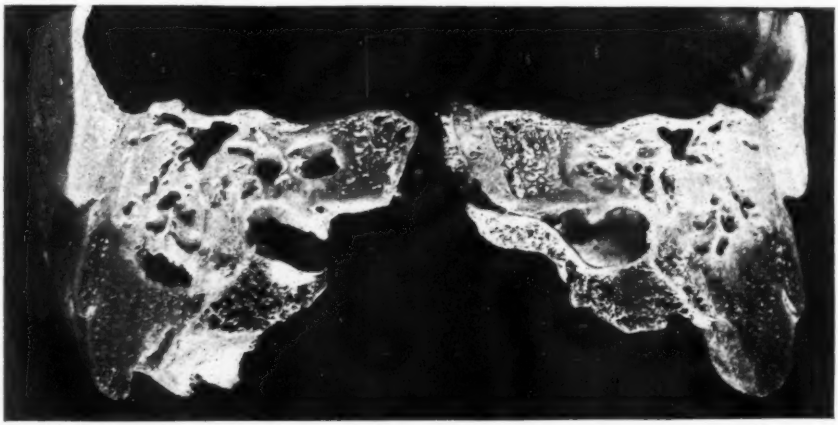


Left.—Outer antral wall diploetic, the diploe separated from the cells (fetal) lining the outer antral wall by a thin layer of compact bone. Mastoid entirely diploetic. The diploe of the zygomatic, squamous and mastoid elements well marked off from one another. The sulcus jugularis is larger than on the right.

No. 21.—MALE, AGED 37 YEARS.

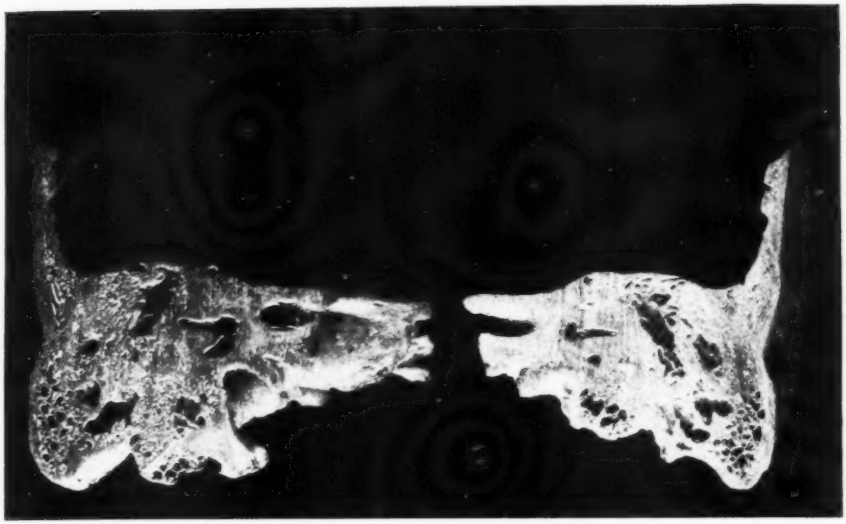


Right.—Outer antral wall dense. A few cells in the upper mastoid. Diploetic lower mastoid. High-lying antrum with an overhanging posterior zygomatic line.

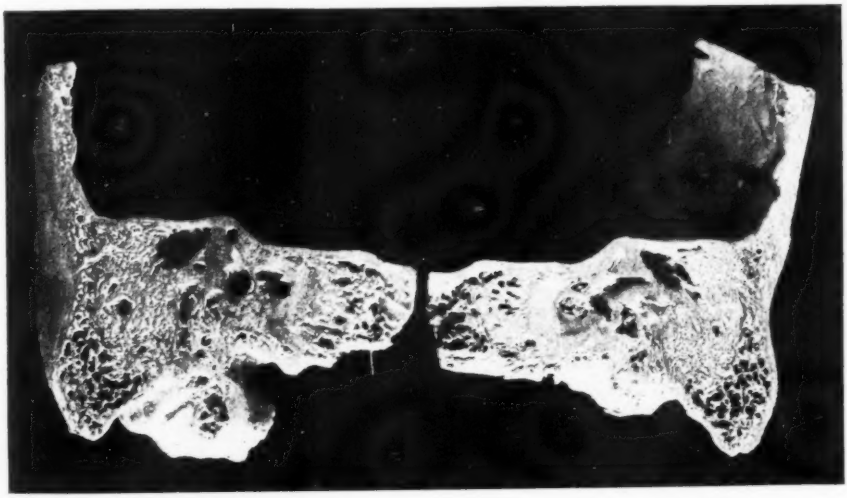


Left.—Outer antral wall has a thin layer of diploe running through it. Cellular upper mastoid. Diploetic lower mastoid. The anterior section shows the marking off of the zygomatic, squamous and petrous elements. High-lying antrum.

No. 22.—FEMALE, AGED 33 YEARS.



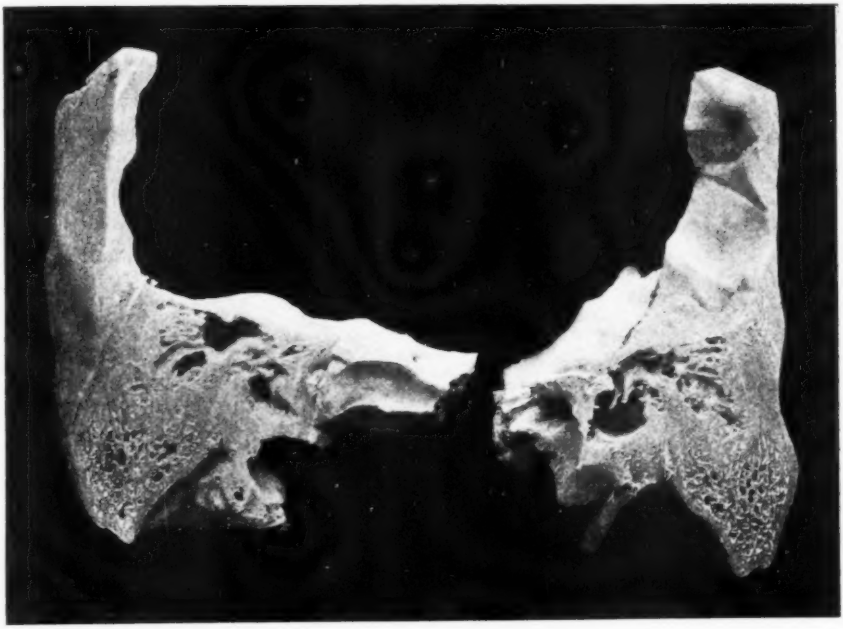
Right.—Outer antral wall dense. A few cells in the inner aspect of the upper mastoid. Diploetic lower mastoid.



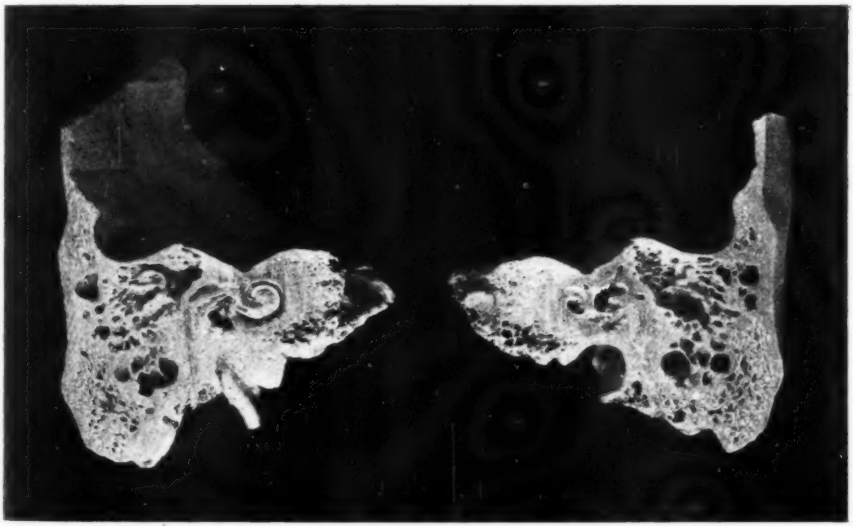
Left.—A thin layer of diploe in the outer antral wall continuous with the diploe of the mastoid. A few cells in the upper part of the upper mastoid.

No. 23.—MALE, AGED 39 YEARS.

Gross asymmetry.



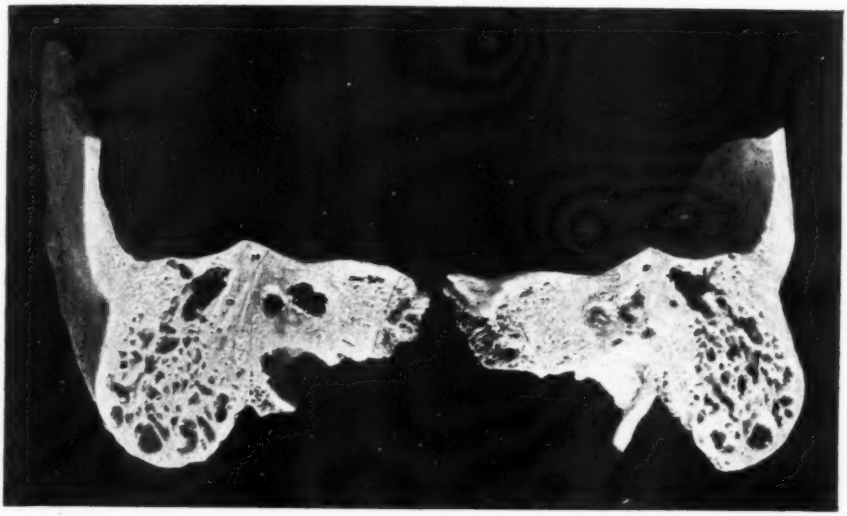
Right.—Diploetic infantile type.



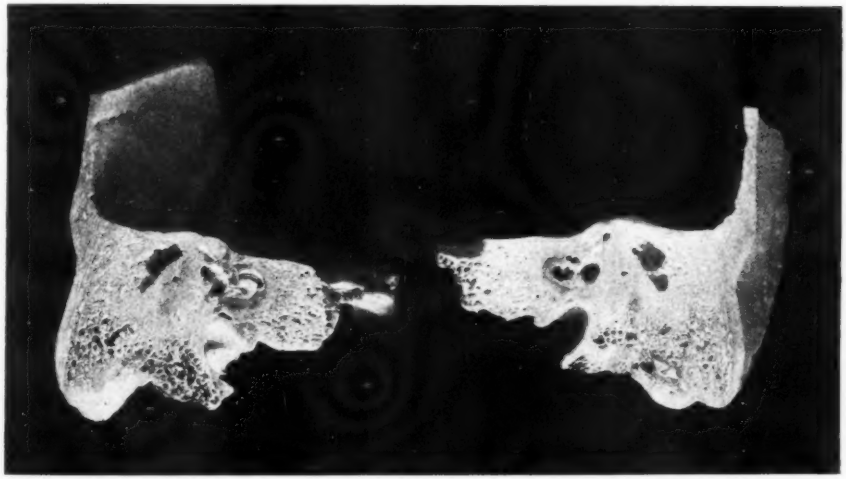
Left.—Outer antral wall and upper mastoid cellular. Lower mastoid diploetic. Dipping down of the middle fossa, causing a sloping roof to the antrum, a condition not seen on the right side.

No. 24.—FEMALE, AGED 41 YEARS.

Gross asymmetry.



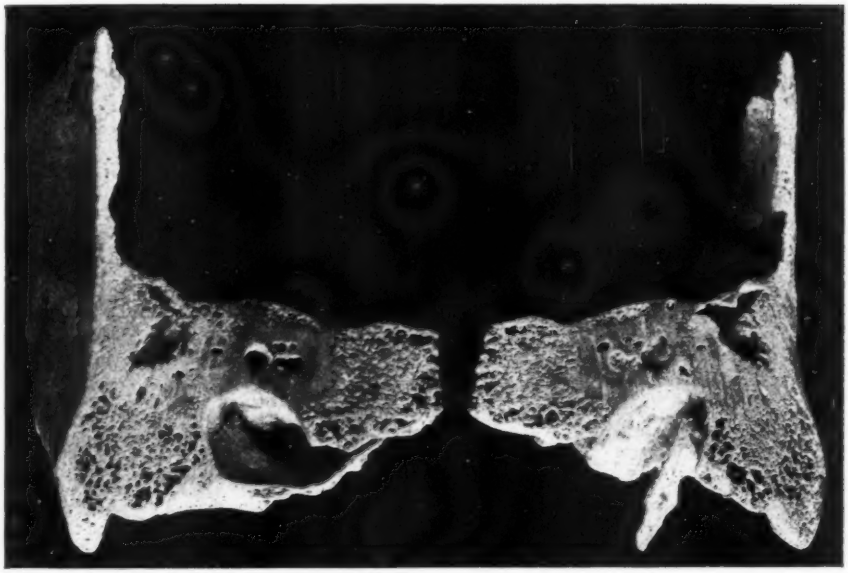
Right.—Outer antral wall dense. Mastoid finely cellular throughout.



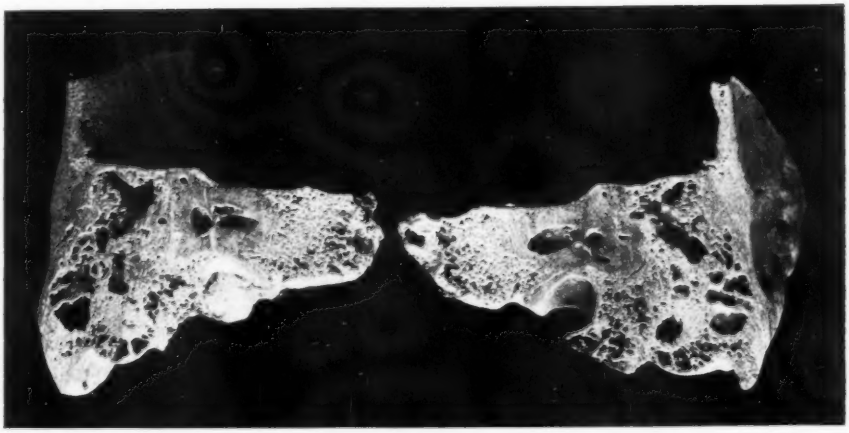
Left.—Diploetic infantile type.

No. 25.—FEMALE, AGED 48 YEARS.

Gross asymmetry.



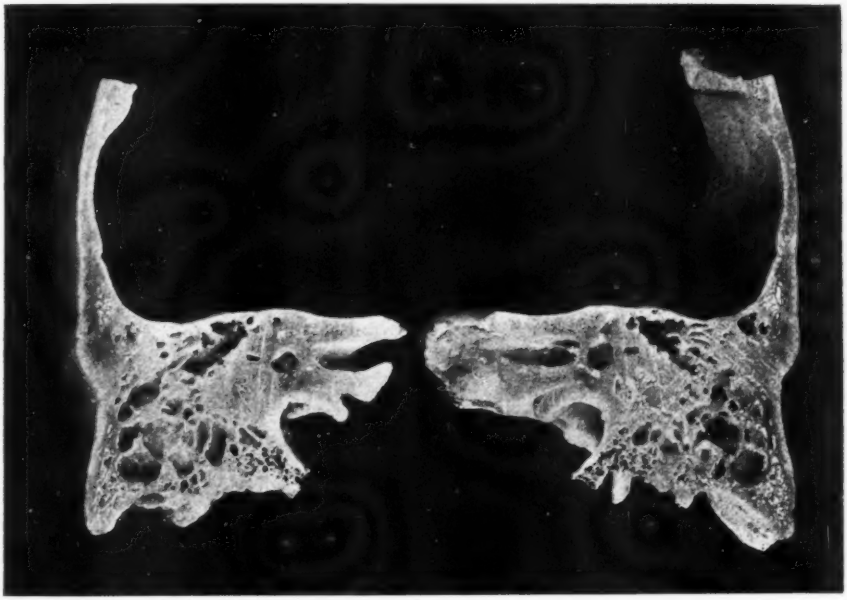
Right.—Diploetic infantile type.



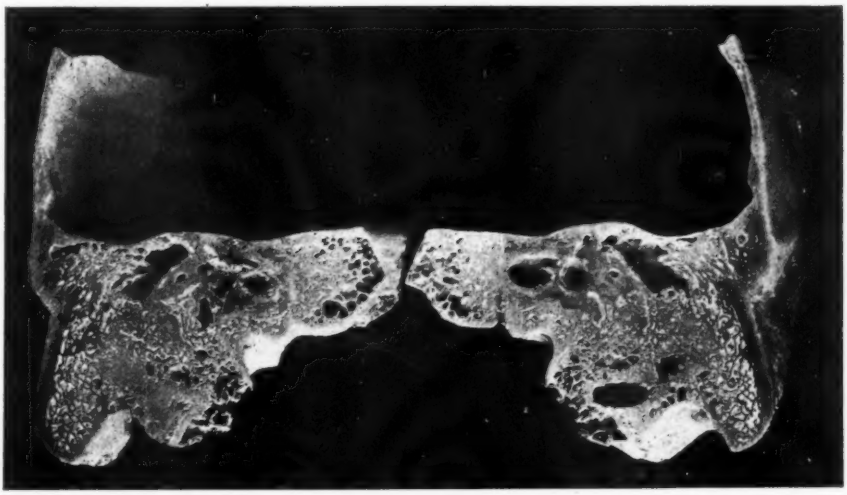
Left.—Dense outer antral wall. Cellular upper mastoid. Diploetic lower mastoid.

No. 26.—MALE, AGED 49 YEARS.

Gross asymmetry



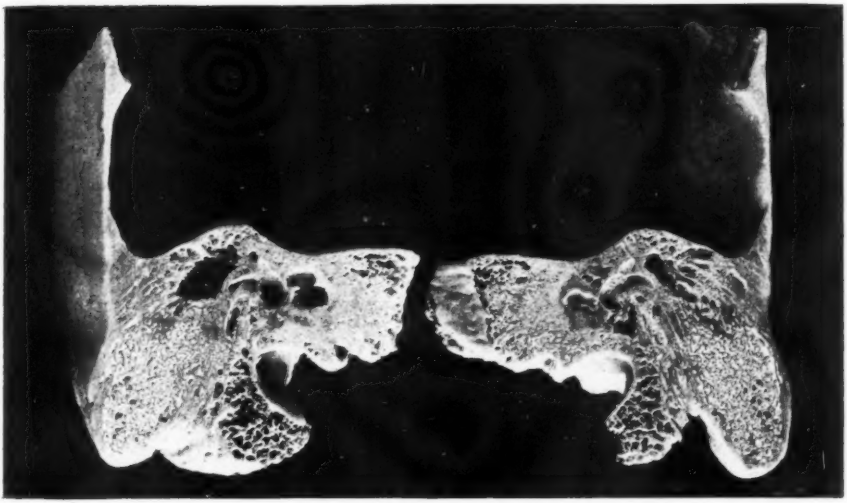
Right.—Outer antral wall dense. Cell in zygomatic diploe. Upper mastoid cellular. Lower mastoid diploetic.



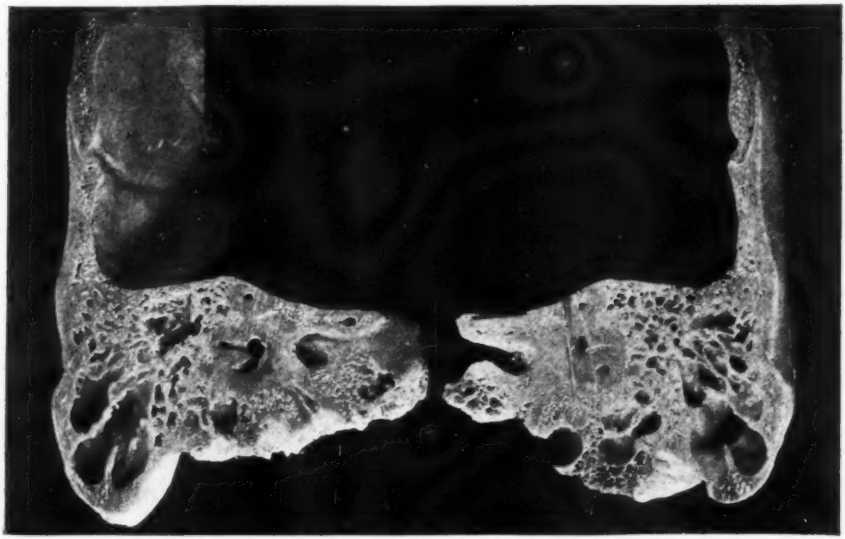
Left.—Diploetic infantile type.

No. 27.—FEMALE, AGED 50 YEARS.

Gross asymmetry.



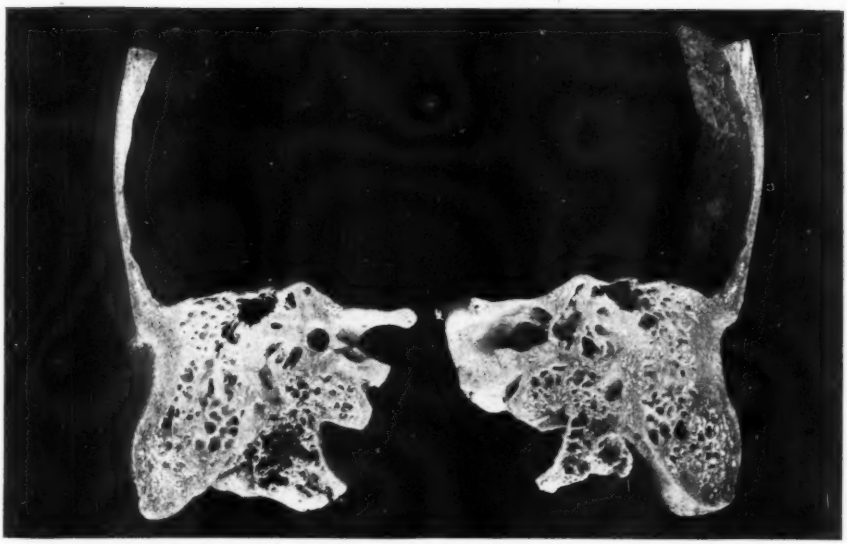
Right.—Diploetic infantile type. Middle fossa dips down, causing a sloping roof to the antrum, a condition not seen on the left side.



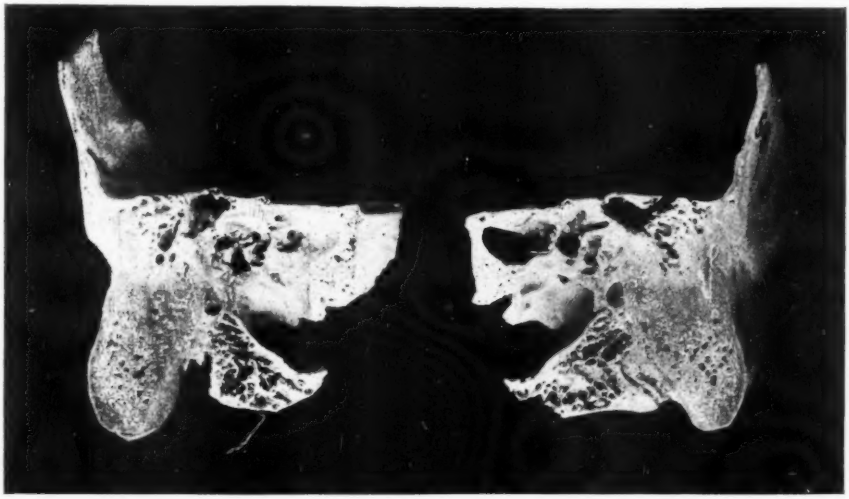
Left.—Cellular outer antral wall and mastoid with a slight rim of diploe at the tip.

No. 28.—MALE, AGED 59 YEARS.

Asymmetry.



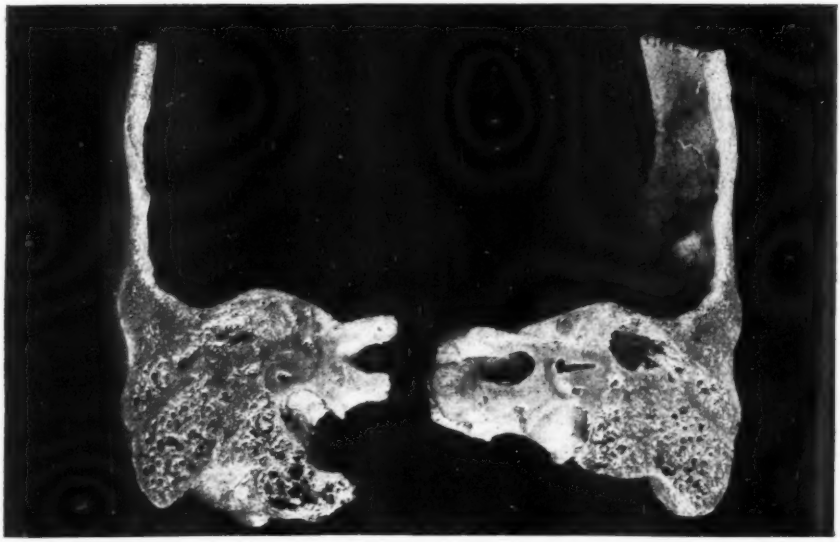
Right.—Outer antral wall partly dense and partly cellular. Fine cells in the upper mastoid. Diploetic lower mastoid.



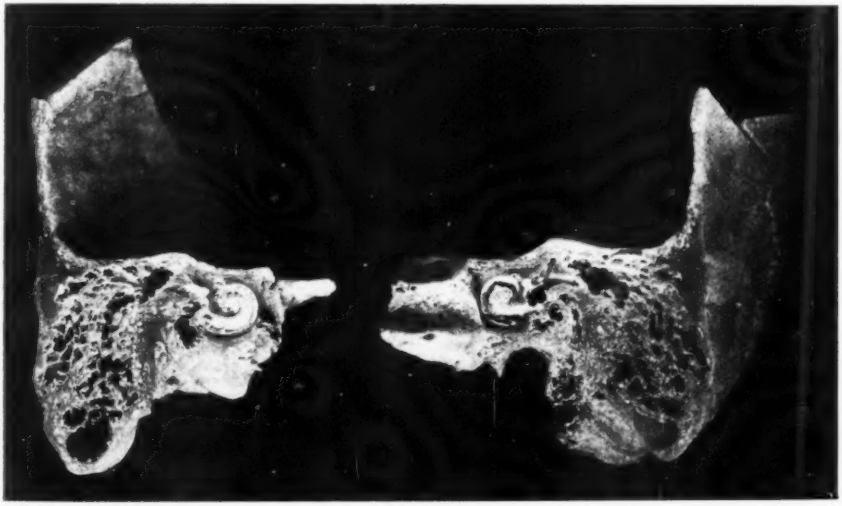
Left.—Diploetic infantile type.

No. 29.—FEMALE, AGED 61 YEARS.

Gross asymmetry.



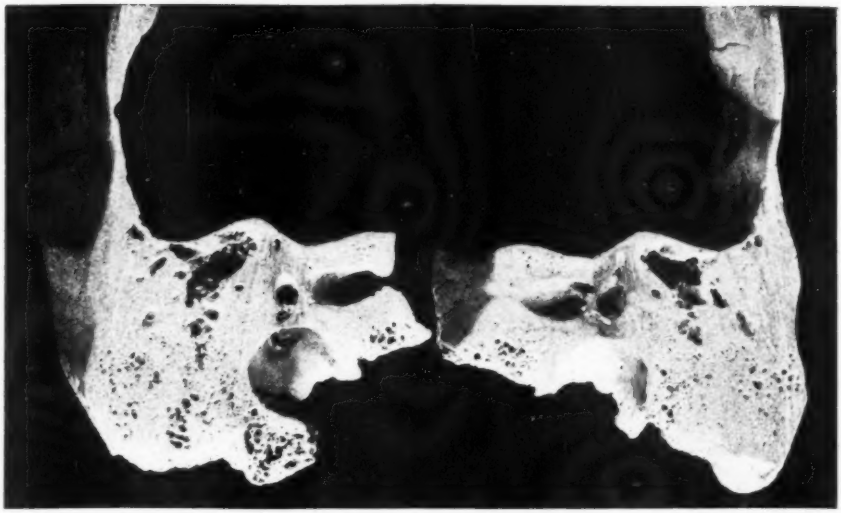
Right.—Diploetic infantile type. The squamous is plainly marked off from the petrous element of the mastoid.



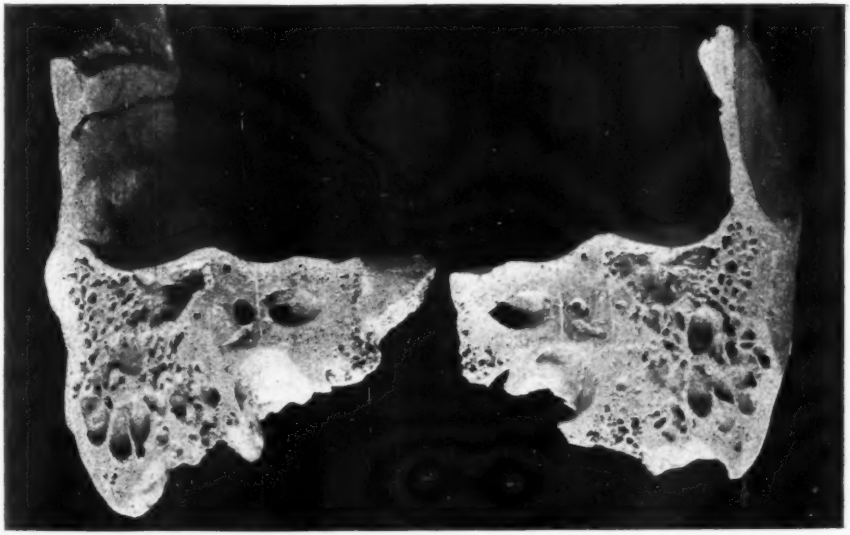
Left.—Cellular outer antral wall and entire mastoid. The elements are also well marked off.

No. 30.—FEMALE, AGED 73 YEARS.

Gross asymmetry.

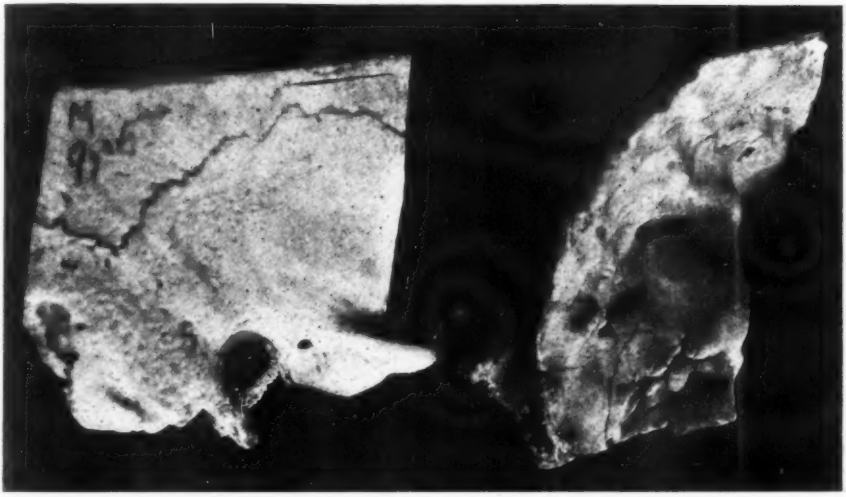


Right.—Diploetic infantile type. The diploe is very dense.



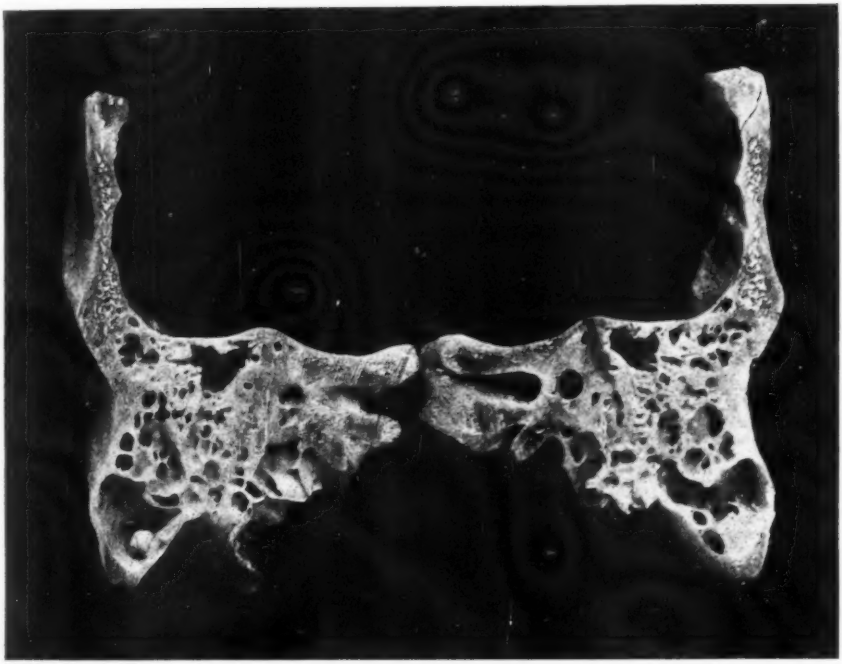
Left.—Outer antral wall and upper mastoid cellular. Lower mastoid diploetic.

No. 31.—MALE, AGED 9 YEARS.



Right Bone.—Showing an external opening for the petrosquamosal sinus in the base of the zygoma on the left fragment. The grooving along the inferior surface on the right fragment. The opening and grooving were absent on the left side.

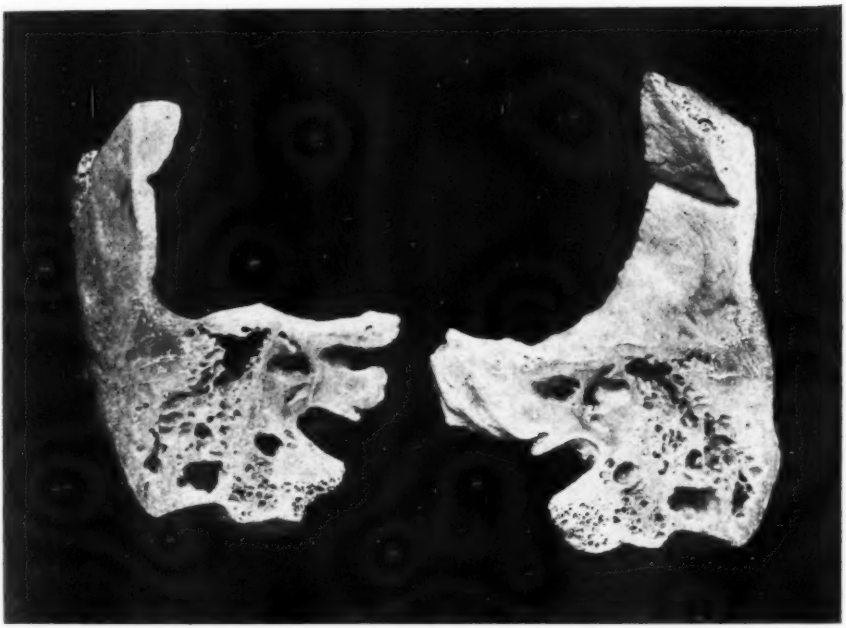
No. 32.—MALE, AGED 59 YEARS.



Right Bone.—A pedunculated exostosis is present, projecting from the inner wall of a large lower mastoid cell.

No. 33.—FEMALE, AGED 61 YEARS.

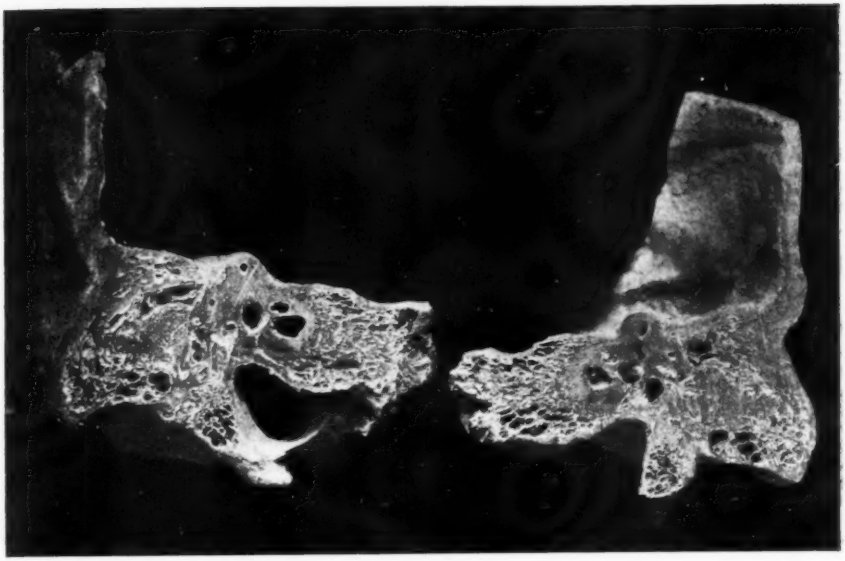
Malformation of the external bony semicircular canal.



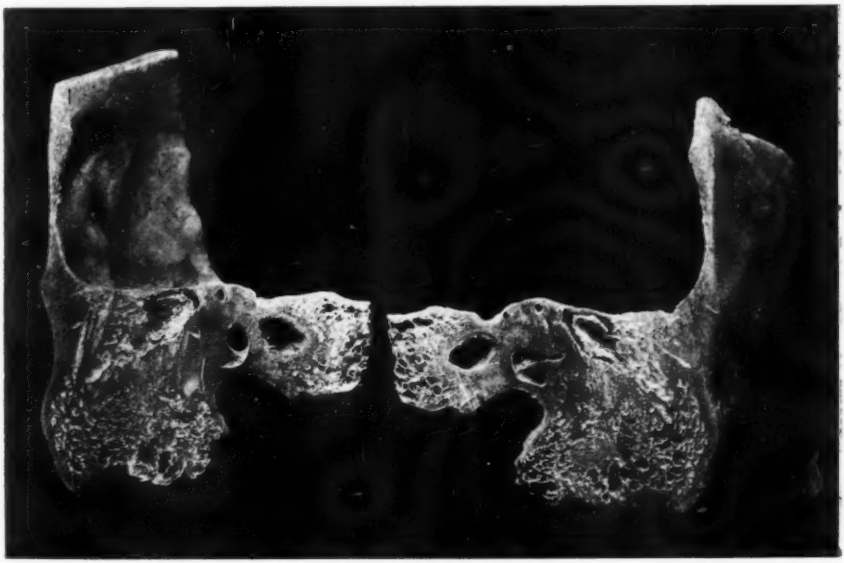
Right Bone.—The canal is represented by a wide cavity opening widely into the outer wall of the vestibule. A small nipple-shaped mass of bone projects from the back part of the sloping roof, and is the only attempt to make the ordinary partitions. The other parts of the labyrinth are normal, and so is the left labyrinth.

No. 34.—MALE, AGED 39 YEARS.

Symmetrical disease in symmetrical bones.

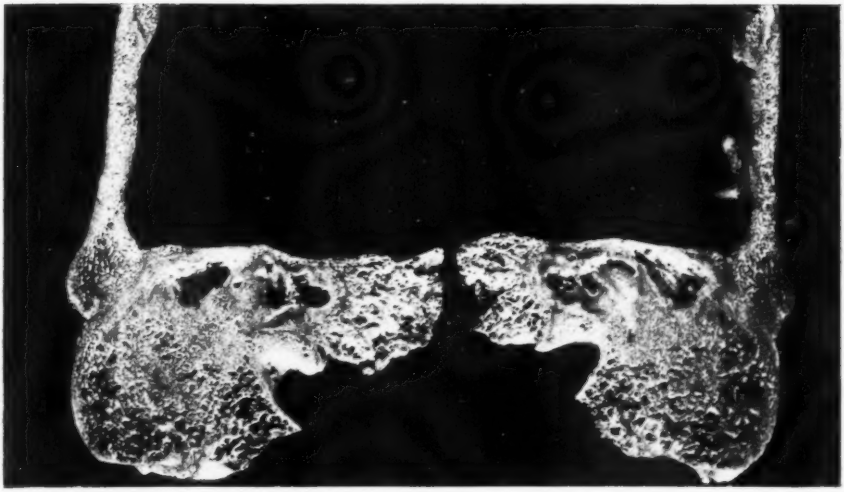


Right.—Chronic middle ear suppuration. Perforation in Shrapnell's membrane. Cholesteatoma filling the antrum. Diploetic infantile type.

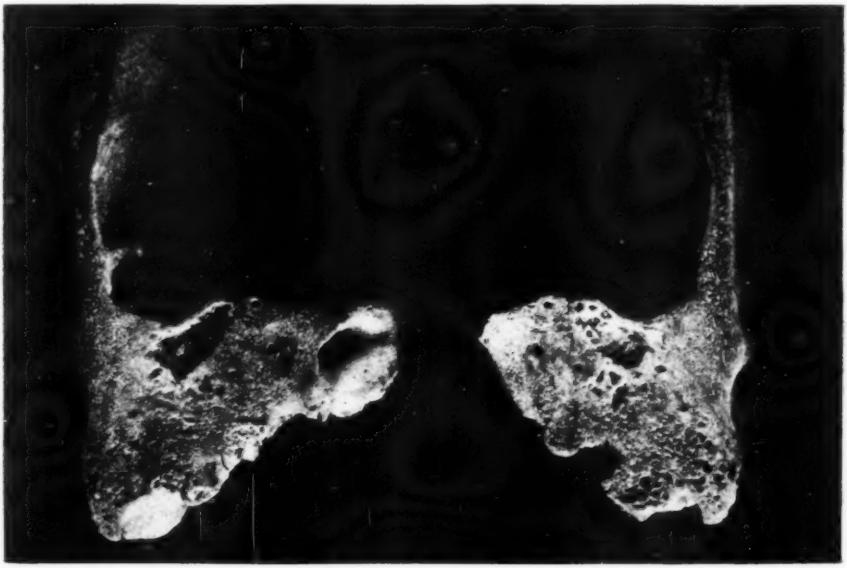


Left.—Symmetrical with the right in type and disease.

No. 35.—FEMALE, AGED 77 YEARS.

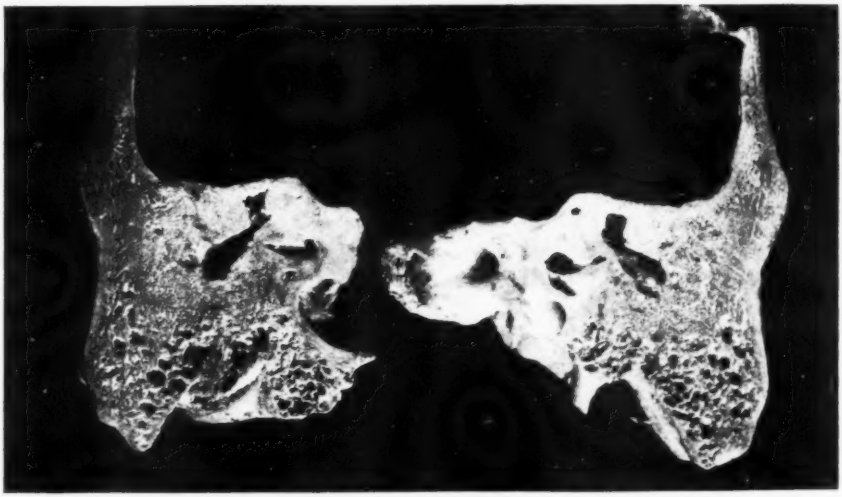


Right.—Diploetic infantile type. Chronic middle ear suppuration. Pus was present in the antrum. The fetal cells lining the outer antral wall are quite destroyed and the antral walls smoothed out. The aditus is much narrowed by osteosclerosis of the roof and fresh bone formation at the apex.

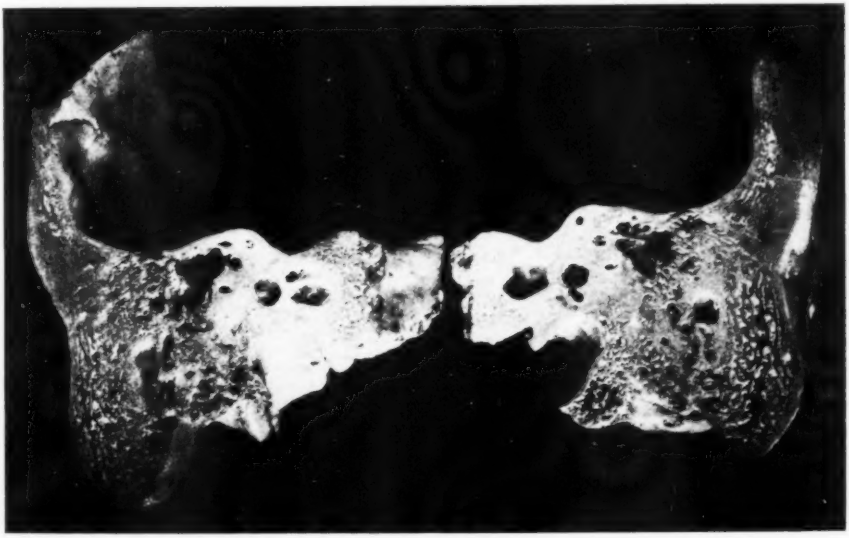


Left.—Diploetic infantile type normal. Intact membrane and ossicles. The middle fossa dips down, causing a sloping roof to the antrum, a condition not present on the right side.

No. 36.—MALE, AGED 49 YEARS.



Right.—Diploetic infantile type. Chronic middle ear suppuration. Pus was present in the antrum, which is much narrowed by osteosclerosis of the fetal cells lining the outer antral wall, the osteosclerotic condition is well marked off from the normal dense outer antral wall which is usual in this type of bone. The antral walls are smoothed out.

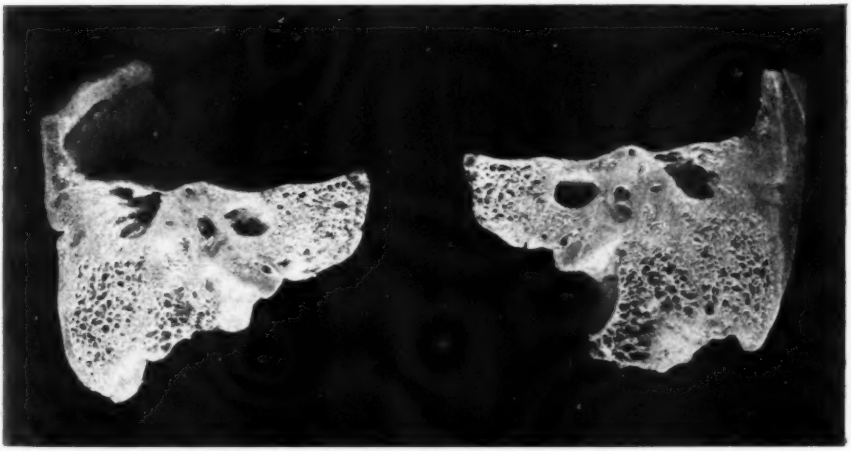


Left.—The outer antral wall is dense and the mastoid mostly diploetic, a few cells run downwards from the apex of the antrum. The membrane and ossicles are intact.

No. 37.—FEMALE, AGED 36 YEARS.

Fixation of the stapes.

Right.—Diploetic infantile type. Bony fixation of the stapes.



Left.—Symmetrical with the right in type and disease.

III.

REPORT OF A CASE OF BRONCHOSCOPY FOR MULTIPLE FOREIGN BODIES (ALMOND SHELL AND PULP) IN A CHILD TWO YEARS OF AGE, WITH SOME OBSERVATIONS UPON BRONCHOSCOPY IN INFANTS AND YOUNG CHILDREN.

By J. R. Winslow, B. A., M. D.,

Baltimore.

On January 5, 1911, at 2 p. m., in accordance with a telegraphic appointment, I met Dr. F. G. Wright, of Chambersburg, Pa., at the University Hospital, in consultation upon the case whose history follows:

Iona B., aged 2 years, was playing upon the floor two days previously, when her mother's attention was attracted by her crying, and she noticed that the child was blue in the face and breathing badly. She immediately held the child up by its feet, slapped her back and running her finger down the throat removed a large amount of almond shell and pulp; this resulted in greatly improved respiration, and the mother supposed that the nut had all been removed.

Since this time, however, the child has at times exhibited embarrassed respiration and occasionally cyanosis.

Examination.—Respiratory movements much shallower on left side and respiratory sounds lost below the second rib, no rales present; temperature, 98 2-5°; pulse, 118; respiration, 28. Slight supraclavicular retraction on left. Right lung normal.

The patient was admitted to the University Hospital and the larynx examined under cocain with the direct Jackson speculum. Nothing was seen in the glottic or subglottic space, and realizing the impossibility of passing a bronchoscope through a larynx of such size without undue force, tracheotomy was determined upon.

Meanwhile a radiograph was obtained, which, as was to be

expected from the nature of the object (nut), revealed no foreign body, but only enlarged peribronchial glands.

On January 6, 1911, at 3 p. m., assisted by Dr. F. G. Wright, I performed a low tracheotomy, under chloroform anesthesia.

Immediately afterwards, with Dr. H. C. Davis in charge of the patient's head and the bronchoscopes, I passed a 7 mm. Jackson tube into the left lower lobe bronchus, which was systematically examined; the tube was, however, too large to enter the upper lobe bronchus. A considerable amount of milky pulp was found in the bronchus and removed with mops; no shell could be discovered.

I had expected to employ suction with Killian's aspirator, in the hope of aspirating the material from the smaller bronchus, but the patient became cyanotic, the pulse weak, and oxygen and amyl nitrite had to be administered.

The bronchoscopic examination was of necessity discontinued, having occupied about thirty minutes.

The tracheotomy wound was left open, long sutures were inserted in the lips, and the nurse instructed to pull the wound open should the patient cough.

The following day (January 7th) the temperature shot up to 103.4° F., respiration 140. Embarrassed respiration, diminished resonance with loss of inspiratory and expiratory murmur below the second rib were present on left side; tubular breathing was heard at left apex.

Subsequently marked cough with expectoration developed. In short, the patient manifested well marked pneumonia.

At this period Dr. C. W. McElfresh was called in consultation and placed in charge of the medical treatment of the case.

From the 9th to the 15th the patient went through the varying phases of a typical pneumonia.

On January 13th the tracheotomy tube, which had been inserted the day following the operation, was permanently removed and the patient was able to breathe through the mouth readily.

On January 17th, the thirteenth day, the patient was discharged from the hospital to return home; temperature 97.3°, pulse 110, respiration 24. Tracheotomy wound nearly closed, general condition much improved. The subsequent history of the case is derived from two letters sent by Dr. Wright. The first, dated February 16, 1911, reports:

Dear Doctor:—The child is still alive, but very ill. When she first came home she was very well, except a running ear and a solid patch a little larger than a silver dollar in the left apex. It seemed as though the trouble would subside and the foreign body become encapsulated, but she developed an influenza (every one here has it) and after a week of coughing developed a pneumonia involving at least the whole of the upper left lobe. After having the pneumonia one week the scar in the trachea opened spontaneously and I opened the skin, allowing a free discharge. The next day she coughed up an oval piece of almond kernel about 6x3 mm. That is four or five days ago, and she is slightly improving. If she gets well will send full data. WRIGHT.

An extract from the second letter, dated February 29, 1912, gives the final outcome of the case:

Dr. John Winslow.

Dear Doctor:—Today I examined Iona B. Sorry we did not report earlier, but they had moved. Have only lately located them. The child is apparently perfectly well, with no signs to show where the trouble in the chest was.

The scar in the neck is rather broad, but the trachea seems solid. After a slow convalescence she recovered fully.

Very truly yours,

FAIRFAX G. WRIGHT.

A more unfavorable case could scarcely be imagined than this one, presenting many difficulties.

1. The child's age (two years) and underdevelopment.

2. The nature of the foreign body, a pulpified nut, furnishing multiple particles which were spattered all over the lung surface, and doubtless entered every bronchiole.

3. The bronchus involved, the left, the most difficult to examine.

I have hesitated whether to call this a successful or an unsuccessful case; while technically I did not succeed in removing all of the foreign body by bronchoscopic methods, yet I am firmly convinced that had the nut-pulp not been removed from the main bronchus, tracheotomy alone would not have enabled the child to clear its lung and survive the first pneumonia.

From a life-saving standpoint the case was most successful,

and one in whose outcome every one concerned has reason to feel gratified.

The pathologic condition in such a case is well illustrated in that reported by F. E. Hopkins (Trans. Am. Lar. Ass'n, 1911).

A female child, four years of age, inhaled a peanut; careful bronchoscopic examination at two sittings failed to reveal a foreign body and the child died on the second day.

"Autopsy showed many (24) small fragments of nuts scattered throughout the lungs and around each a pneumonic area.

"Instead of a single nut occluding the trachea or larger bronchi, the many fragments of the well-chewed nut were shot into the smaller bronchi."

Thomas Hubbard reports similar autopsy findings (ibid.) in a peanut case, in his practice.

Cases of foreign bodies in the lungs of young children and infants (say of four years and under) are coming under our care in increasing numbers, because through the writings of Killian, Jackson, Ingals, Coolidge, Halsted, Mosher, Hubbard and other masters of bronchoscopy, the general profession is being educated to recognize these conditions and their proper method of treatment.

These cases constitute the most difficult in the whole field of bronchoscopy, both on account of the small size of the respiratory passages at this period of life and the difficulties of instrumental manipulation, as well as the nature of the objects usually encountered.

While a young child is liable to place almost anything in the mouth and inhale it, owing to the small size of the glottis (6 mm. infants, Jackson—7 mm. three years, Killian) large objects cannot pass through into the lower passages, therefore many of the foreign bodies commonest in adults are rarely found in the lungs of very young children (pieces of bone), while those encountered belong to the class most difficult of removal, small or multiple objects (nut shells, pulp, beads, pins, beans, pebbles, etc.).

The younger the child the greater the difficulty and urgency, and the mortality is high, despite successful removal. So that it has seemed to me that these cases constitute a group of themselves well worthy of the discussion of this representative body, and for this purpose I have brought the subject before you.

What is our best course of procedure in this class of cases? Should we tracheotomize at once, as the primary procedure, or has the advent of bronchoscopy largely abolished the necessity for this operation?

The desirability of upper bronchoscopy (without tracheotomy) is obvious, and it should be the routine method were there no disadvantages associated with it.

In prebronchoscopic days tracheotomy was the method of choice, whose success is attested, among numerous others, by the remarkable series of four cases of foreign bodies in the bronchi of small children under two and one-half years of age, reported by our fellow T. H. Halsted (Trans. Am. L. R. O. Soc., 1902).

In these young children upper bronchoscopy has serious objections attached to it. The anatomic structures are small, rendering the manipulation of instruments difficult and resulting in loss of time and irritation of tissues.

A study of the cases reported shows that while upper bronchoscopy has been frequently attempted for the removal of foreign bodies in infants, in a large percentage, if not the majority of the cases, tracheotomy has become ultimately necessary for successful removal.

Now, if such be the situation, why not tracheotomize at once and operate by the easier and more certain route (lower bronchoscopy)?

In a recent article (*Deutsch. med. Wochenschrift*, June 29, 1911) G. Killian has made a most valuable contribution, in which he calls attention to another aspect of this subject, namely, the frequency with which tracheotomy or intubation becomes necessary after upper bronchoscopy, even when successful, reporting a series of nineteen cases under seven years of age, some of them personal and some derived from literature. He also cites a series of thirty-five cases reported by Schneider, of Moscow, of which five required intubation or tracheotomy. He concludes that these procedures stand in a causal relation to upper bronchoscopy, inasmuch as the changes necessitating them occur within a relatively short period (6 to 37 hours) afterward; that the site of the change is the subglottic space, as evidenced by the stridor and the results of intubation or tracheotomy, and in a few cases by direct or indirect laryngeal examination.

We know from both clinical experience and postmortem evidence that inflammatory swellings are prone to occur in the subglottic space. Children from the seventh, and especially from the fourth, year downwards are especially liable to such swellings after upper bronchoscopy. In this connection the relation of the caliber of the tube to the width of the subglottic space becomes of paramount importance. No reliable measurements have hitherto existed. In order to furnish such data, Killian undertook the measurement of twenty-two children's larynges in fresh cadavers. For this purpose the employed a series of semicircular metal sounds of increasing size, observing which one passed the cricoid region easily, which with difficulty, which not at all. In this way he determined that the width of the subglottic space stands in no especial relation to the sex of the child, and in but little to the age, the most important and hitherto neglected moment being the bodily development.

Upon this basis he has constructed the following table:

Body length.	Diameter of subglottic space measured with calibrated metal sounds.	Calculated periphery of the lumen.	Age.
45 cm.	3.5 mm. (4 not)	10.99 mm.	8 mos.
50 "	4.5 mm. (5 not)	14.13 "	4 "
52 "	5 mm.	15.7 "	3.5 "
53 "	4.5 mm. (5 not)	14.13 "	3 "
53 "	5.5 mm.	17.27 "	newborn
53 "	4.5 mm. (5 not)	14.13 "	" "
56 "	4.75 mm.	14.91 "	?
58 "	5 mm. (5.5 pressure)	15.7 "	2.5 mos.
58 "	5.5 mm.	17.27 "	4 "
60 "	5.5 mm.	17.27 "	9.5 "
62 "	5.5 mm. (6 with difficulty)	17.27 "	6 "
62 "	6 mm. (6.5 not)	18.84 "	18 "
63 "	5.5 mm.	17.27 "	9 "
64 "	5.5 mm.	17.27 "	13 "
65 "	6.5 mm. (7 not)	20.41 "	8 "
70 "	5 mm.	15.7 "	16 "
80 "	6.5 mm. (scarcely)	20.41 "	12 "
80 "	6.5 mm. (7 not)	20.41 "	24 "
86 "	7.5 mm. (8 not)	23.44 "	48 "
94 "	7 mm. (7.5 not)	21.98 "	36 "
120 "	8 mm.	25.12 "	7 yrs.
? "	9.5 mm.	29.83 "	10 "

A study of this table shows that the subglottic space is much narrower than is commonly supposed; that it increases gradually with the body length, but not commensurately, and is subject to great variations. Comparison of these measurements with the age of the child, in the series collected, shows that in some of the cases the bronchoscopic tube was too large for the subglottic space, and must have caused trauma.

Killian observes that we still require a great deal of information as to the anatomic relations of the child's larynx at different ages and different grades of bodily development, and promises to take this up for future research.

The selection of method must be individual, depending upon a number of considerations.

1. Age of the patient: as Killian has demonstrated, this is more a matter of physical development than of age: nationality also may have a bearing. At least my intubation experiences have shown me that in certain nationalities (Italian) the larynx is smaller than the corresponding age.

Some years ago Ingals announced that he had rarely found upper bronchoscopy satisfactory in children under three years of age.

Recently Finder, apropos to an unsuccessful case of a piece of bone in the right bronchus of an eleven months old child, reported to the Berlin Laryngological Society, stated that henceforth he will resort to inferior bronchoscopy in all children in the first year of life.

In discussing this case E. Mayer went still further and considers inferior bronchoscopy preferable in children six years of age and under. Brünings advises it in all children under two years of age, as a routine procedure.

Nehrkorn recommends low bronchoscopy in all young children.

Jackson, however, regards tracheotomy as being "unnecessary nine times out of ten, and believes that it should be limited to dyspneic cases." I should like to know whether he intends this to apply to these very young children.

2. The nature of the foreign body is of great importance in determining this question.

Objects which are liable to swell so that they cannot be withdrawn through the subglottic space (beans) should be removed by the lower route (Nehrkorn, Killian).

Likewise brittle objects which are liable to be broken into several fragments, or multiple objects necessitating a great deal of manipulation; objects which are irritating in themselves and certain to be followed by pulmonary inflammation (peanut shells, pepper corns), should be removed without irritation of the subglottic space by instruments.

3. The duration of the condition is of a determining moment: the presence of a foreign body rapidly produces a catarrhal condition of the air passages in children, increasing the vulnerability of the mucous membrane to instrumental manipulation; therefore, when the foreign body has remained for a long time, tracheotomy is indicated (E. Mayer, Killian).

4. The side affected must be considered, for it is much more difficult to remove a foreign body from the left bronchus, especially by upper bronchoscopy. Among thirteen cases in children collected by Killian, in nearly two-thirds the foreign body was found in the left bronchus.

5. The condition of the subglottic space is of paramount importance: this should always be determined by direct or indirect laryngeal examination before undertaking upper bronchoscopy.

Any outspoken subglottic swelling constitutes a contraindication to upper bronchoscopy.

The passage of the bronchoscopic tube will inevitably result in the necessity for tracheotomy; it is therefore better to perform this operation primarily and employ lower bronchoscopy.

In conclusion, it seems to me that the present situation with regard to foreign bodies in the lungs of young children has been well summarized in the advice of Hubbard, "When in doubt do tracheotomy."

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IV.

TUMORS OF THE LARYNX.*

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It is not the purpose of this paper to present an exhaustive treatise upon laryngeal tumors, but rather to call attention to a number of practical points in the operative treatment and to emphasize these points by illustrative cases.

Although intralaryngeal tumors were probably recognized and removed prior to the advent of the laryngoscope, it was not until this instrument came into use that their recognition or removal was attended with any degree of accuracy. Within a few years of Garcia's discovery the first operation for laryngeal neoplasm was performed. The question of priority, as in many other subjects, is a vexed one. Wright gives Lewin credit for the first intralaryngeal operation for the removal of a laryngeal growth under the guidance of the mirror, which he is said to have performed on July 20, 1860, while Jurasz credits Victor von Bruns with the first operation, performed in 1861, and does not even mention Lewin. From these dates the number of tumors observed and extirpated by this method grew with great rapidity, and the scarcity of the growths of recent years has been ascribed to the avidity with which they were removed in the early days of laryngoscopy. As Wright so well expresses it, "no subject so immediately engaged attention as that of laryngeal tumors, forming striking pictures in the laryngeal mirror, causing marked and distressing symptoms, capable of immediate relief by means of instruments under the guidance of the laryngoscope, and last, but by no means least, affording the operator a chance to display in the most brilliant manner his newly acquired skill."

The removal of tumors from the larynx per vias naturales and under the guidance of the laryngoscope may be termed the

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laryngoscopic method, to distinguish it from more recent intralaryngeal manipulation by means of endoscopic instruments.

For the successful laryngoscopic operation proper illumination is absolutely essential. The source of light must be brilliant, white, uniform and free from conflicting shadows. These qualities are best obtained by using the Nernst electric light placed in a suitably constructed metal cylinder.

The instrument table should be conveniently placed to the operator's left, and should contain, besides the instruments needed, gauze sponges, sterile napkins, spirit lamp, etc. Three assistants are desirable. The duties of the first assistant are most important, and consist in holding the patient's tongue, if it is found that the patient cannot do this himself, in controlling the epiglottis and in manipulating intralaryngeal instruments under the direction of the operator, should it be found necessary to use more than the one in the hand of operator himself. A second assistant presides over the instrument table, and a third holds the patient's head and retains the mouth gag in position if it becomes necessary to use one. At times it is necessary to pass a silk ligature through the tip of the tongue, instead of holding the tongue with a napkin. This is particularly serviceable in operating under general anesthesia.

One of the greatest obstacles to satisfactory endolaryngeal operation is a depressed epiglottis, especially under general anesthesia. To control this the device described by Horsford, in which a properly constructed needle holder enables one to pass a ligature through the free border of the epiglottis, is recommended. This ligature can then be held by the first assistant or grasped by a pair of hemostat forceps and allowed to hang out of the mouth. Pfau's modification of Horsford's instrument (Figure 1) makes it easier of application in many cases. By its use a needle properly threaded is placed in one of its blades, which, when closed upon the opposite blade, permits the needle to be grasped and withdrawn with the instrument, the ligature remaining in the tissue.

An additional aid to laryngoscopic operation, as indeed it is to careful examinations, is the magnifying anastigmatic mirror of Brüning, which enlarges the laryngeal picture two and one-quarter times.

Anesthesia.—Endolaryngeal operations may be conducted under local or general anesthesia.

Local Anesthesia.—Since the advent of cocain, operations within the larynx have become greatly simplified, and many of the previous difficulties in the way of preliminary training have been almost completely overcome. The proper local application of cocain or some of its substitutes is productive of uniformly satisfactory anesthesia.

Cocain remains the most reliable drug, but owing to its toxic properties, betaeucain may be substituted at times. Where unsatisfactory results are obtained one can usually determine a faulty technic in its application. In a few rare instances the use of this local anesthetic in quantity approaching the danger line has failed to anesthetize the mucous membrane of the larynx, so that the slightest touch of the instrument has resulted in a violent exhibition of the laryngeal reflex. There is a distinct difference between the retention of sensation and the reflex contraction of muscles. In these cases local anesthesia may fail.

Cocain poisoning is less likely to occur if it is so applied that little if any is swallowed by the patient. Its application directly to the field of operation is therefore desirable. Reflex contraction may be avoided by applying the anesthetic to the soft palate, posterior pharyngeal wall and posterior surface of the epiglottis. This being a large surface to cover, a solution may be sprayed upon the parts as a preliminary procedure. If cocain is used, it should not be more than a 2 per cent solution. The immediate effect of this is to cause, in addition to anesthesia, a most disagreeable and, to some individuals, alarming sensation of constriction. The dangers of poisoning are much greater where solutions are used in spray than where the remedy is applied by cotton applicator. The addition of a solution of one of the adrenalin preparations in the strength of 1/8000 will inhibit the cocain intoxication. This may be almost completely avoided, however, by substituting a 2 per cent to 4 per cent solution of betaeucain. It has been shown that this drug is very much less toxic than cocain, and while its anesthetic properties may not be so pronounced or reliable, it generally answers for the preliminary spraying. After waiting five minutes the field of operation should be carefully painted with a 20 per cent solution of cocain by means of the cotton applicator. This should be thoroughly moistened with the solution.

but not to the extent of permitting any of it to drip from the cotton. Repeated applications of the stronger solution should be made at intervals of two minutes until one determines by touching the parts that anesthesia is complete. The sensation of the patient of local constriction may also be a guide.

In a certain proportion of cases the application by means of the cotton swab is ineffectual. Here five drops of the 20 per cent solution may be applied directly to the field by means of the laryngeal syringe. This should, of course, be preceded by the preliminary spraying with the weak solution.

The submucous injection of cocain solution for anesthesia has been practiced by Herying and others. This is particularly recommended for those cases in which swollen or infiltrated masses are to be extirpated, such as are to be found in tuberculosis. The technic of this method is greatly facilitated by the use of Chappel's syringe. (Figure 2.) Having produced preliminary anesthesia, the needle of this instrument is pressed well into the submucous tissue and by a slight movement of the thumb the spring releases the piston, forcing the fluid into the parts. The advantage of this instrument is in the accuracy with which it can be used. Anesthesia by this method is more rapidly produced and persists for a somewhat longer time than by the method of topical application.

The method of inducing local anesthesia by injection into the superior laryngeal nerve has been described by Frey and others, and recommended especially for the relief of painful affections of the larynx. It may, however, be satisfactorily utilized in the intralaryngeal operations, especially in excessive irritability of the upper half of the larynx. Twenty drops of a 1 per cent solution of cocain in combination with 1/1000 solution of adrenalin gives satisfactory results. The success of this method depends upon waiting the proper length of time after the injection before beginning intralaryngeal manipulation. In some cases anesthesia is complete in three minutes; in others, twenty minutes must be allowed to elapse.

General Anesthesia.—In relatively few instances does it become necessary to administer a general anesthetic for endolaryngeal operations, except in cases of children or young adults. In the author's experience the adolescent age is particularly resistant to satisfactory local anesthesia of the larynx.

When general anesthesia is necessary, the choice of anesthetic becomes a question of considerable importance. Generally speaking, ether is to be preferred, although its well known irritating effect upon the mucous membrane of the upper air passages, producing excessive secretion, is a decided disadvantage. On the other hand, the element of safety enters as a more important factor, and particularly where the almost erect or sitting position of the patient becomes a necessity. Chloroform, while producing ideal anesthesia and specially useful in children, adds greatly to the dangers and compels one to keep the patient in a reclining or semirecumbent position, a most difficult and at times impossible position for intralaryngeal operations.

The excessive secretion in ether anesthesia may be greatly diminished by the administration of atropin one hour before the operation.

In using general anesthesia the patient should be placed in an operating chair so constructed that he may be raised to a sitting posture or lowered to the recumbent position at will. Anesthesia should be begun in the recumbent position, and when complete the patient should be gradually raised to the desired position. A chair is preferable to an operating table, in that it permits the operator to sit directly in front of the patient, and not to either side, which places him at an inconvenient angle for ease and accuracy of manipulation. In the case of young children the anesthetic may be begun upon a table, the patient being lifted to the lap of a nurse before commencing the operation.

Complete surgical anesthesia is absolutely essential. It is extremely difficult to continue the administration of the anesthetic during the operation. This may be partially accomplished either by holding a gauze sponge saturated with the anesthetic and grasped by a long pair of forceps in the vicinity of the nose and mouth, or by the use of a properly constructed tube, similar to that of the Junker inhaler, placed in the right angle of the mouth. As a rule, this will not interfere with the use of the laryngoscope, and is out of the way of laryngeal forceps or other operating instruments.

It is well known that the laryngeal reflex is one of the last to disappear under anesthesia. Even when profound narcosis

has been established, the moment an instrument is introduced into the larynx, reflex spasm occurs. In order to overcome this, a solution of cocain and adrenalin should be applied, especially over the posterior surface of the epiglottis. This can best be done by means of the cotton applicator. The strength of the solution should vary with the age of the child. Young children do not bear cocain well. A 1 per cent to 2 per cent solution in combination with a 1-10,000 adrenalin solution, answers every purpose. In young adults stronger solutions may be used, generally 5 per cent being sufficient. If this does not answer, the percentage may be increased to 15 per cent or 20 per cent, care being observed to have the cotton moist but not dripping.

Before proceeding with the description of operative methods, a brief consideration of the relative value of endolaryngeal and external operations may not be out of place.

Although the laryngoscopic method is less applicable to carcinoma, even here it may be of value. It has been advocated by Gouguenheim and Lombard:

First—In certain varieties of intrinsic cancer which remain limited for a long time, showing no tendency to spread.

Second—In old people whose advanced age would make us hesitate before proposing a large surgical intervention, and

Third—In certain pedunculated epitheliomata, the rapid removal of which is necessary to prevent suffocation.

In malignant disease the most generally useful purpose, after all, to which intralaryngeal surgery may be put is for that of diagnosis. The operation of removing a section may be performed with impunity in all cases. The caution so frequently given, that unless the patient be prepared for a radical operation the growth should not be attacked, is only of minor importance. It is absolutely essential to confirm the clinical diagnosis by histologic examination. If this examination proves negative, no harm has been done, and the way is clear for further and possibly successful treatment. If this examination proves positive, the growth may or may not take on rapid growth. Should the patient refuse more radical operation and the tumor rapidly progress, the fatal termination may be somewhat advanced; this, however, must be looked upon as of much less importance

than the question of an early confirmed diagnosis, which must remain, as previously stated, the saving factor in a possible cure. If the malignant growth has already assumed large proportions and has invaded surrounding glands, the clinical diagnosis usually suffices. The tumor may have been of very slow growth, and it is much wiser to leave it alone rather than to encourage its growth by removing a section to confirm a diagnosis which needs no confirmation. What has been said above with regard to removing a piece for diagnostic purposes, applies only to small circumscribed tumors of doubtful diagnosis.

The two varieties of malignant growths found in the larynx, as elsewhere, are sarcoma and carcinoma. For many years the relative value of endolaryngeal and extralaryngeal operations for these neoplasms has been a matter of much discussion. the burden of which has had reference to carcinoma. Sarcoma has been given comparatively little consideration, evidently due to the fact that it is of relatively rare occurrence. The fact that sarcoma is less likely to spread to adjacent structures, especially in its early stages remaining circumscribed and confined, favors the endolaryngeal method somewhat more in this variety of malignant growth than in carcinoma. Laryngologists have from the beginning leaned naturally to the laryngoscopic method. Unfavorable results, however, have compelled the conclusion that in but few instances is it possible to completely eradicate a malignant growth by removing it through the natural passages. A few brilliant results have been reported, especially by Fränkel, who among others has been the most ardent advocate of this method. Statistics have been of comparatively little value, for, as Jurasz has pointed out, the value of statistics depends upon more careful differentiation of the period or state of development of the growth at which the operation is undertaken. Cases of cure by both methods have been reported, the early diagnosis being in all instances the important factor. The improved technic of today renders it possible to obtain results by radical external operations which were impossible a few years ago. At that time, while it was possible to completely extirpate a growth by radical measures, the gravity of the operation was so great that the attempt was generally made to remove the mass by simpler procedures. Today, however, less

is to be feared from the operation than from the rapid development and extension of the malignant growth, and therefore those cases which were formerly attacked by endolaryngeal methods, in the hope of avoiding more serious operation, and were followed by indifferent results, are today more generally and more successfully removed by laryngectomy or laryngotomy.

Nevertheless, endolaryngeal procedures in malignant growths may find their uses.

Generally speaking, all benign tumors should be removed through the natural passages rather than by external incision. The size and position of the tumor are the most important determining factors in its removal. Supraglottic tumors, infraglottic tumors, providing they do not extend too far downward, and even intratracheal growths, may be extirpated under the guidance of the laryngoscope. The question of choosing between this method and the direct endoscopic procedure is not as yet capable of decision. Owing to the large number of laryngologists trained in the use of the mirror, compared to the few whose technical skill enables them to use easily the direct method, the former will doubtless for many years be the method of choice. It must be conceded, also, that patients are more readily trained to the required manipulation of the laryngoscopic method than they are to the method by the direct laryngeal speculum, which offers another reason for adopting the former. On the other hand, it is selfevident that owing to the situation of a tumor, it may be inaccessible except by direct laryngoscopy. This is peculiarly true of growths situated low in the larynx or in the trachea. The trained laryngologist may proceed with safety by first attempting the laryngoscopic method, failing in which he may then adopt the direct method.

In children, multiple papillomata are fairly frequent. Here the removal of these growths by the endoscopic method is often the procedure of choice. As a rule, whatever operation is undertaken in children, general anesthesia is necessary. The laryngeal speculum of Mosher and the tracheoscopic and bronchoscopic tubes of Killian and Jackson have made the removal of papillomata in children comparatively simple. For the removal of these tumors small, straight, cutting forceps or the spiral instrument devised by Mosher is recommended.

A method of suspension laryngoscopy recently described by Killian, although requiring elaborate apparatus, will probably add materially to the ease of intralaryngeal manipulations by the direct method. By the use of this suspension apparatus the operator brings the laryngeal cavity into the field of vision, holding it there, and leaving his two hands free for other manipulations.

Among other recognized measures for the removal of papillomata in children, it should be remembered that tracheotomy and intubation have both their advocates. It is a well recognized fact that no matter what operation or how frequently it is performed, papillomata recur with exasperating rapidity. It is also well recognized that these tumors have a life history, and that after a certain length of time they not only cease their growth, but retrogress. In undertaking operations for their removal, therefore, one is fortunate if he chooses the psychologic moment, that is to say, the particular time when the tumor has reached its maturity. In children the presence of papillomatous growths frequently causes alarming dyspnea. Tracheotomy performed at this time places the larynx at rest, following which the natural tendency for retrogression of these tumors asserts itself and their spontaneous disappearance frequently results. In one of the author's cases treated in this manner, a tracheotomy tube was worn for over two years. Various intralaryngeal manipulations had been unsuccessfully tried. The case made a complete recovery, after all attempts at removing the tumors had been discontinued, recovering with a good voice, by spontaneous disappearance of the growths.

In a certain proportion of cases in children in which the tumors do not develop at the entrance of the larynx, but confine their growth to the vocal bands and surrounding structures, intubation has been successfully used. Not only does the intubation relieve dyspnea, thus placing the larynx at rest, but the intubation tube causes a certain amount of gentle and continuous pressure which may have a beneficial effect. At any rate, the writer has successfully treated cases in this way, the last one being a child three years of age, in whom the intubation tube was worn almost continuously from November 7, 1910, to December 8, 1911, eleven months, with the exception of about four months, during which time the breathing was

good. Becoming worse after a severe cold, reintubation was necessary. The child has now been without a tube about ten months, and when last heard from was breathing well and had had but slightly husky voice.

It is the author's practice to remove the tube at intervals of from three to six weeks, cleanse it and reintubate immediately, if necessary, or within a very short time. In some cases calcareous deposits form around the metallic tube which become a source of irritation and even cause superficial ulcerations. A new tube must be used whenever the one removed shows any sign of roughness.

The treatment of multiple papillomata of the larynx in children by fulguration, although not as yet found in medical literature, has been practiced by Dr. Harmon Smith of New York, and probably others. Dr. Smith's experience has been more extensive than other operators. In a personal communication dated September 19th, Dr. Smith states, "that fulguration will cut down these growths, but they soon return, and at the present writing I am unable to say that it has any features of a definite character beyond the fact that it removes the growths easily and prevents the necessity of tracheotomy." Dr. Smith's method is to apply the spark for a few seconds until the surface becomes white. The operation is performed under chloroform and cocain anesthesia and with the Jackson tube.

Besides the methods already mentioned, which are, of course, performed without the use of the laryngoscope, those under the mirror's guidance are by means of chemical caustics, galvanocautery, electrolysis, the sponge method of Voltolini, incision, forceps, the cold or galvanocautery snare and the guillotine.

Chemical caustics may be used in small nodules, small papillomata, pachydermia and tuberculous or syphilitic excrescences. They are of value in that form of multiple papillomata in which the neoplasm seems to take on the nature of diffused papillomatous infiltration. This condition is well illustrated in Figure 3. In this patient a large number of papillomata were removed by the forceps until it was found difficult to remove more than very minute sections. Here repeated cauterization proved beneficial. The best method of using chemical caustics is by fusing a crystal of nitrate of silver or chromic acid upon a

suitably curved applicator, or inserting a crystal of chromic acid in the fenestrum of a hollow applicator, such as is used for carrying trichloracetic acid. (Figure 4.)

The galvanocautery is effective where delicacy of application is desired. It is particularly useful for the removal of small vascular growths and varicosities. After the removal of angiomata there frequently remains a small vascular point, which may be the beginning of a new growth, and which is best removed by the galvanocautery point.

Electrolysis is applicable to tumors with broad sessile bases as well as to those in which hemorrhage is liable to be an important factor. Fibromata, cystomata, chondromata and certain angiomata may be attacked in this manner. In some instances malignant tumors have been known to diminish in size by the use of electrolysis. This at best can only be a makeshift.

Figure 5 illustrates a variety of tumor readily treated by incision. The tumor proved to be, as its appearance indicated, a cystoma. Cystomata are nearly always broad at their attachments, and in many instances it is impossible to grasp them by means of the ordinary forceps in use. One of the numerous laryngeal knives, especially that devised by Herying, may be used. In this case incision evacuated a small quantity of thick opaque fluid. The sac disappeared and there was no recurrence.

The removal of tumors by laryngeal forceps is the most generally applicable of all methods. Many varieties of these instruments have been devised, and tumors have been removed by evulsion, by crushing and by abscision. It is generally conceded that cutting forceps are the most desirable, their action being more precise as well as more easily controlled. They should be so constructed that all portions of the larynx may be readily reached by permitting movements from below upwards as well as anterposterior and lateral. These qualities may be found in the Mackenzie or Scheinmann instruments. Among the best of the cutting forceps are those known as Cordes, which have also been called double curettes. Their action is from below upwards, and may be applied with great delicacy to almost any position in the larynx. Figures 6, 7, 8 show a fibroangioma which was readily grasped by a Mackenzie forceps and removed by evulsion. The bleeding that followed was

somewhat more profuse than ordinarily, but was not alarming. The action of the Cordes forceps is well illustrated by Figures 9, 10, 11, showing a fibroma of the left vocal band.

The galvanocautery snare is particularly applicable to bleeding tumors; it also may be used for removing portions of neoplasms which cannot be removed in toto.

All tumors, of whatever nature, presenting any degree of pedunculated attachments, may be readily removed by the cold wire snare. Even tumors that have a sessile base may be attacked in this way, providing the projecting portion of the neoplasm is not smaller than the base.

Tumors springing from the epiglottis are readily surrounded by the snare wire. The most frequent variety of tumors found here are the lipoma and the cystoma. The snare is much more applicable to the former than to the latter. In applying the snare to tumors in this region, the straight variety used for operation within the nose, or that possessing a slight curve, may be easily applied. The method of procedure is slightly modified over that used for ordinary intralaryngeal operations. The modification consists in depressing the tongue instead of drawing it forward. For this purpose a tongue depressor with a long handle is desirable, the patient being instructed to hold the instrument in position himself. The laryngoscopic mirror in such case may be entirely discarded.

Tumors situated below the epiglottis, whether large or small, may be reached by the snare without much difficulty, providing they are slightly pedunculated. Among the numerous instruments devised for this purpose, the Schroetter snare, as improved by Coakley, is one of the best. The wire used should be sufficiently firm to remain in whatever position it is placed. It should be of fair strength, although this is of less importance than in other snare operations, such as are performed in the nose or for the removal of tonsils. Number 6 piano wire is small, strong and sufficiently stiff for all purposes. It should be threaded to the snare in such a manner that the loop rests in an anteroposterior direction for tumors situated in the anterior or posterior portions of the larynx. By introducing the ends of the wire into the eyes of the stylet from opposite sides, or both ends from the same side, one may obtain the two positions of the loop as desired. The loop should be large enough to

readily surround the neoplasm. It should not, however, be too large, in which case it becomes difficult to enter the larynx with it, and also for the reason that the manipulation of drawing the wire tightly over the growth is thus prolonged.

In removing a tumor by this method the snare is accurately applied over the tumor mass, then by gentle movements to and fro, or from side to side, depending upon the position of the tumor, it is brought as close to the point of attachment as possible. The wire is now carefully drawn taut and gentle traction is made to determine whether the tumor is held well in the grasp of the wire. If the tumor does not slip out of the loop, one is assured that the position of the instrument is correct. The next step in the procedure consists in removing the growth, and this is best accomplished by making gentle traction at the same time that the wire is drawn through. By this method the tumor is removed by a combination of cutting and pulling.

The entire operation can be performed in a few seconds; much shorter time, in fact, than is required to describe it.

For the removal of large tumors of the larynx, the usual method of using the snare should be modified. It becomes difficult and at times impossible to introduce a wire snare in such a manner that it will completely surround the entire mass, especially if but little space remains unoccupied by the growth. This difficulty may be satisfactorily overcome by proceeding in the following manner: Through the loop of wire one passes first a pair of laryngeal forceps, the handle of which may be locked. An assistant holds steadily the snare while the operator, passing the forceps into the larynx, grasps the tumor and locks them. The forceps are now passed to an assistant, the operator exchanging them for the snare. The tumor being held firmly by the forceps, it becomes a simple matter to pass the snare loop completely over the growth. This step may be facilitated by instructing the assistant to make gentle traction, thus drawing the tumor into the loop at the same time that the operator passes the loop around the tumor. (Figures 12, 13.) Having completely surrounded the neoplasm in this manner, the rest of the procedure is accomplished in the same manner that other snare operations are performed, as described above. In a large pedunculated carcinoma, situated in the opening of the larynx and attached to the right aryepiglottic fold and

ventricular band, the author succeeded with great ease in removing the tumor en masse. Previous attempts at surrounding the tumor with the wire in the ordinary method had failed, owing to its size and position. (Figure 14.) This case was of unusual interest, inasmuch as its diagnosis was uncertain, even after pathologic examination. After removal there still remained an unexplainable infiltration, which was inconsistent with benign neoplasms. Subsequent developments proved the malignant nature of the growth.

Pedunculated carcinomata of the larynx are extremely infrequent. Two other cases have been reported, one by Ballenger and the other by Fraenkel.

Fig. 1.—Pfau-Horsford epiglottis suture needle.

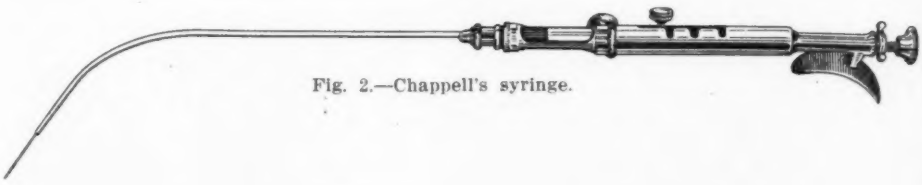

Fig. 2.—Chappell's syringe.

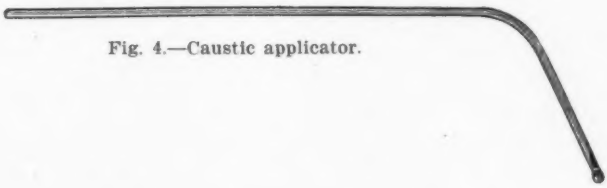

Fig. 4.—Caustic applicator.

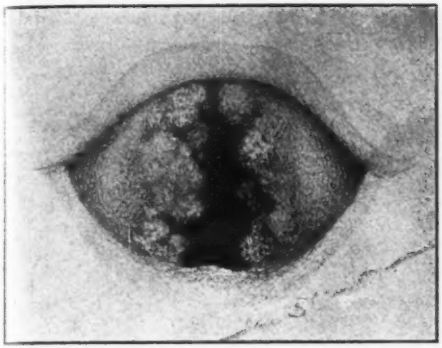

Fig. 3.—Multiple papillomata with papillomatous infiltration.

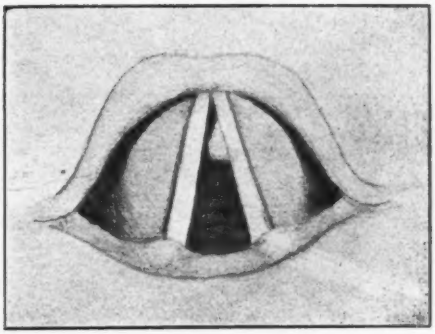

Fig. 5.—Cystoma removed by incision.

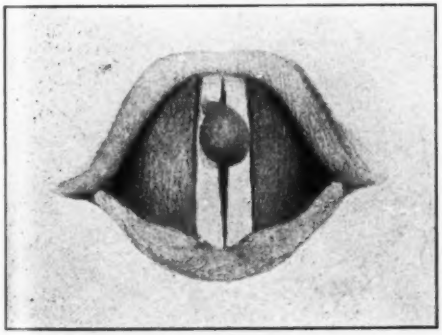

Fig. 6.—Fibroangioma, pedunculated and freely movable during respiration. Showing position of tumor during phonation.

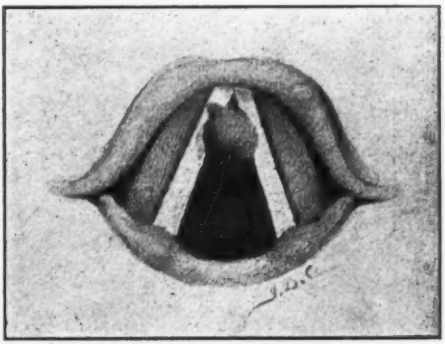

Fig. 7.—Fibroangioma, showing position of tumor during forced inspiration.

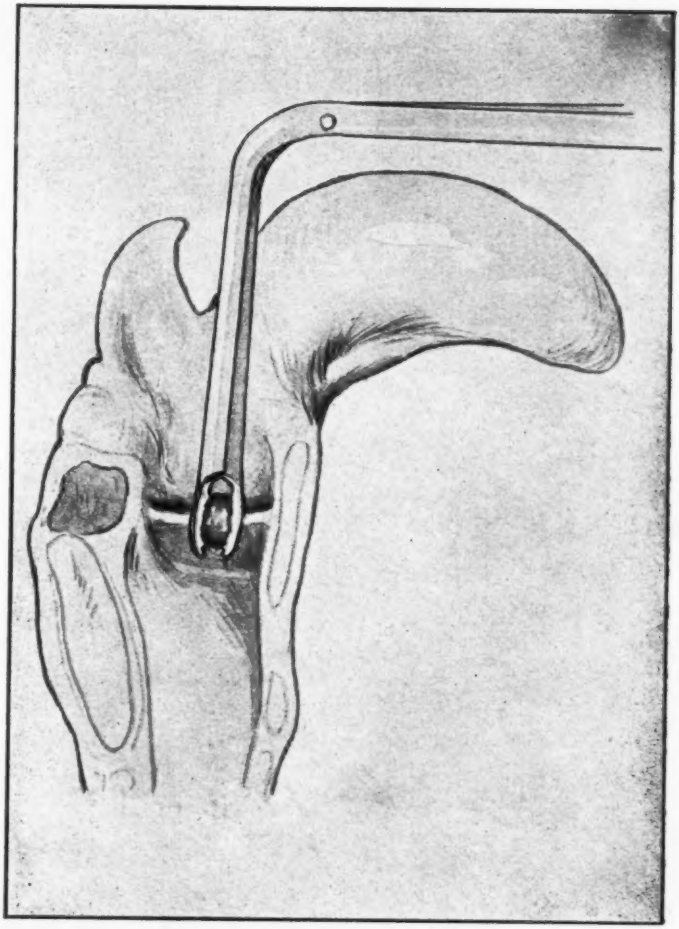

Fig. 8.—Showing removal of movable fibroangioma. Evulsion with Mackenzie forceps.

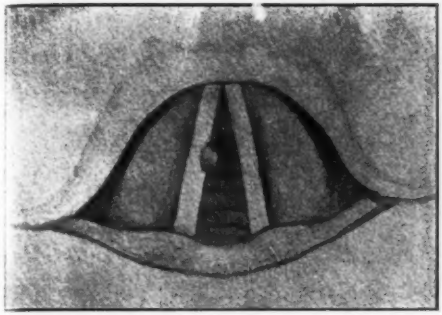

Fig. 9.—Fibroma from left vocal band during inspiration.

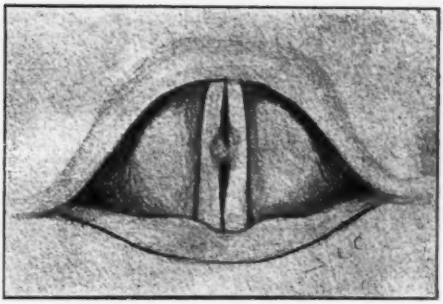

Fig. 10.—Fibroma from left vocal band during phonation.

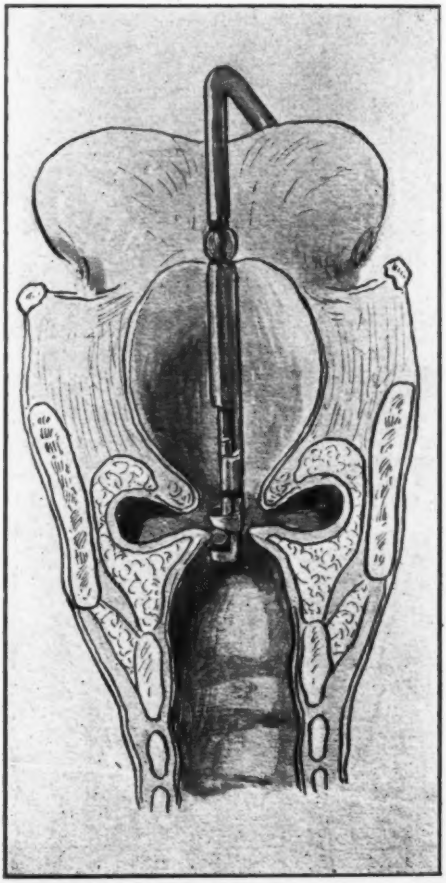

Fig. 11.—Showing removal of fibroma from vocal band by Corde's forceps.

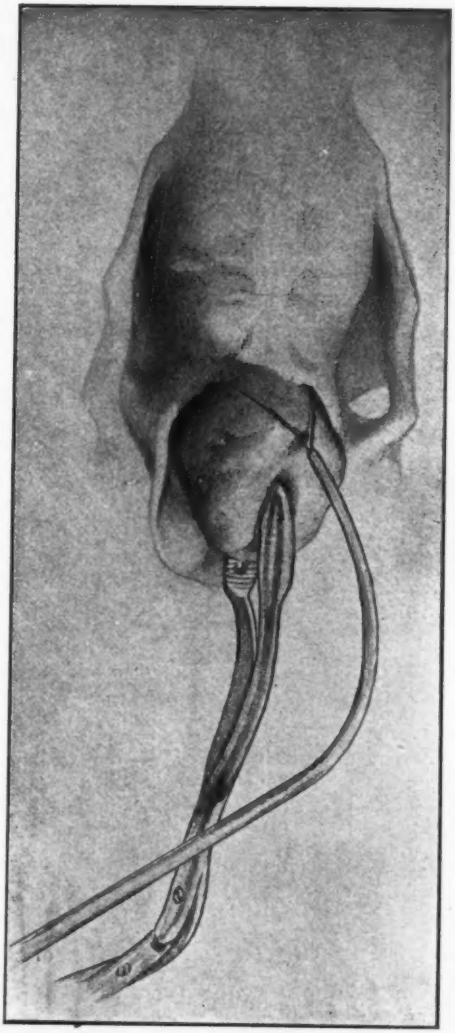

Fig. 13.—Removal of pedunculated laryngeal tumor. Showing snare and forceps in position.

Fig. 12.—Large pedunculated carcinoma of the larynx.

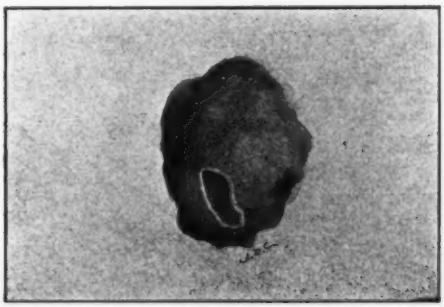

Fig. 14.—Pedunculated laryngeal tumor, life size. Some sections show an overgrowth of epithelium not sufficiently atypical to justify a diagnosis of malignancy. Other sections show small nests containing two and three epithelial cells which led to the diagnosis of carcinoma. The subsequent clinical history confirmed this diasgnosis.

V.

ACUTE SUPPURATION OF THE MIDDLE EAR—ITS NEGLECT AND PROPER TREATMENT.*

By Prof. Dr. Gustav Alexander,

Vienna.

Otitis media may be caused by a great number of different microorganisms. In the cases which have been published, every possible bacterial exciting cause has been found, sometimes in pure culture and sometimes as mixed infections. Saprophytes, also, have occasionally been found in the discharge, but in such cases we seem to have a secondary infection complicating the primary cause of the suppuration.

PATHOLOGIC ANATOMY.

In all cases where the middle ear is infected through the tube, we observe, first, a swelling of the tubal membrane which soon leads to obstruction of the tube. This swelling soon spreads to the mucous membrane of the middle ear and the tympanic cavity, as well as to the entire middle ear, viz., the meso- and hypotympanum, the attic, the antrum, and in many cases the pneumatic cells of the mastoid process become filled with an infectious viscid or hemorrhagic exudate. After a short time, sometimes a few hours only, suppuration is present, and the purulent inflammation of the mucous membrane of the tympanic cavity and drum leads to extensive exudation in the middle ear cavity. The pus finally breaks through the tympanic membrane and drains outward. During the earlier stages of the perforation, the secretion is chiefly of a bloody color, and not until later does it become clear, especially in influenza and typhoid otitis.

If the process runs a normal course, in about one week the secretion becomes a mixture of pus and mucus; the pus be-

*Read by invitation before the British Medical Association, at the eighteenth annual meeting, Liverpool, 1912.

comes ropy and more and more replaced by mucus, owing to the daily decrease of the secretion. The swelling of the mucous membrane of the tympanum and tympanic cavity decreases. The hole in the drum, in normal cases, closes after the secretion has ceased and complete restitutio ad integrum results, both anatomically and functionally. Sometimes in children, after a few weeks, the macerated epidermal crust is thrown off, leaving a fresh, normal, smooth and shining epidermal layer which has formed under it.

SYMPTOMS.

The prominent symptom, especially at the beginning of the disease, is the sudden, spontaneous and severe pain in the ear. This pain is often excruciating, and the patient is restless and sleepless. Sometimes, this severe pain is preceded by a sense of fullness and obstruction in the ear, as well as unilateral headache.

The loss of hearing reaches a high degree (speech, 1-2 m.) in the typical genuine otitis media suppurativa. Towards the end of the disease the hearing returns, and in uncomplicated cases becomes normal after the disease is cured.

At the beginning of the otitis media suppurativa, we usually find considerable fever, sometimes 40 degrees C. and higher. When the discharge of pus takes place, after the perforation of the tympanum, the temperature falls in cases running a normal course. An unfavorable symptom is a sudden, spontaneous fall of the temperature to normal or subnormal in the early stages of the inflammation, unless accompanied by a subsidence of all other symptoms of the disease. This is observed in cases of early intracranial affection, and is sometimes found in individuals lacking power of resistance or weakened by severe and general suffering from otitis media suppurativa. In early childhood, uncomplicated otitis media suppurativa often begins with vomiting and chills.

The condition of the tympanic membrane.—The otoscopic findings are a more or less pronounced swelling and redness of the tympanic membrane, which may involve the entire drum. In some cases, although the entire membrane may be inflamed, certain parts are involved to a greater extent, i. e., deep red color, severe swelling and bulging. Often extensive formation of vessels is observed. When the epitympanum

alone is involved (Kümmell), the inflammatory symptoms are limited to the upper part of the tympanic membrane, i. e., to Schrapnell's membrane and the upper posterior quadrant, and often lead to bulging of the upper posterior part of the drum membrane (Politzer).

The deep red color of the tympanic membrane is by no means a reliable indication of the degree of the otitis media. Sometimes in a simple otitis media, and even in a myringitis, the tympanic membrane is of a deep red color, and greatly bulging. Owing to the bulging, the line of the handle of the hammer is entirely invisible. In cases of otitis media due to influenza, especially, the membrane may be of a deep red color even in very mild cases, and this may be accompanied by an elevation of the epidermis in the form of vesicles. On the other hand, the tympanic membrane may show a pale red color, even with a large collection of pus in the middle ear (empyema), and under certain circumstances may show only a radiate and peripheric vascular injection. In general, the intensity of the redness and bulging of the tympanic membrane decrease slightly with the progress of the purulent softening of the exudate in the middle ear. If the acute otitis media has led to an empyema of the middle ear cavities, especially of the tympanic cavity, without a perforation, the membrane sometimes appears opaque and grayish yellow, and will show only a slight injection of the radiate vessels of the tympanic membrane.

The expected perforation is generally indicated by a circumscribed yellow discoloration and marked bulging of the tympanic membrane. If it has taken place, we see an opening which is more or less round, allowing a free passage to the pus; or it may be a fissure, the edges of which approximate, just allowing the pus to escape. If the secretion is mopped out, it will sometimes be impossible to see the site of the perforation. We usually recognize it by the oozing of the secretion, though it may be ascertained by the use of Siegle's speculum. Sometimes the perforation is situated at the top of the mastoid process.

The size of the perforation varies. In the fulminating type of otitis media suppurativa accompanying acute infectious diseases, especially scarlet fever, we find comparatively large openings and in rare cases extensive destructions.

In advanced stages of suppuration the mucous membrane of the tympanic cavity resembles the tympanic membrane in redness, swelling and moisture. Later, that of the tympanic membrane decreases, and the epidermal layer becomes dry. Now, the perforation in the membrane is distinctly visible, even if such was not the case previously. The margin of the perforation is thicker than the surrounding tympanic membrane, but finally becomes of the same thickness. As soon as the perforation heals, the margin is grayish white.

The test of the function of the ear shows in every case a considerable decrease in sound conduction. At the beginning of the inflammation the sense of hearing is greatly diminished. At this time it is even possible to elicit a slight, spontaneous nystagmus to either side when looking in that direction, evidently in consequence of the pressure due to the secretion. It disappears with the appearance of the perforation.

DIAGNOSIS.

Even in the early stages of the disease the diagnosis of otitis media suppurativa can be made without any special difficulty, in most cases, from the anamnesis and the characteristic symptoms, viz., sudden appearance of violent pains in the ear, difficulty in hearing, fever, and the condition of the tympanic membrane.

From the standpoint of differential diagnosis, only the simple otitis media comes into consideration. Great bulging of the tympanic membrane, teatlike bulging of the upper posterior quadrant, especially circumscribed yellowish discoloration, and great loss of hearing, provided the hearing was normal previously; these symptoms speak against a simple otitis media and in favor of an otitis media suppurativa, as does the continuous pain, which at times becomes very severe. Attacks of pain with intervals free from pain speak more for a simple otitis media. High fever indicates otitis media suppurativa. In children up to the age of eight years, and especially in babies, we are unable to test the sense of hearing objectively and exactly. We may, however, make a test by means of instruments making a loud noise, or by means of sounds familiar to children, e. g., a bell, jangling of keys, sounding glasses and high pitched tuning forks, and thus ascertain approximate-

ly the degree of diminution of hearing. In some cases direct examinaton of the antrum (transillumination, X-ray examination) is of great service.

Little children complain of severe pain which they refer to the affected ear, but which is caused by late dentition, acute inflammation of the pharyngeal lymphatic glands, tonsillitis or the swelling of the pharyngeal tonsils. In some cases intense pains in the ear are an early symptom of an acute infection, usually following an affection of the mucous membrane of the oropharynx. At the time of puberty, violent pains in the ear sometimes indicate the beginning of an otosclerosis, if the otoscopic findings are negative.

In the stage of suppuration the odorless, profuse, pulsating and purulent secretion, and later on the odorless mucopus, speak for otitis media purulenta as opposed to otitis externa. If at the same time we find furunculosis of the external auditory meatus (otitis externa furunculosa), we may find it difficult at the first examination to make a diagnosis of whether we have to deal with a simple inflammation of the meatus, or with one complicated by an otitis media suppurativa, especially if we find multiple furunculosis of the external auditory canal with considerable purulent secretion.

In such cases the differential diagnosis is made on the basis of the following points:

(1) There is no fever in otitis externa of older children and adults; in small children we sometimes observe a moderate rise in temperature. If, therefore, in doubtful cases the anamnesis shows that the ear disease started with a high fever, we may assume, also, the existence of an otitis media.

(2) If in a case of otitis externa we cleanse the meatus and make a passageway by inserting a thin ear speculum, we will obtain a normal or slightly diminished aural acuity, if the drum is not too thickly covered with epidermis. If, however, there also exists an otitis media suppurativa, the hearing will not be improved after cleansing the external auditory canal.

(3) In children of four years or older, the bony auditory canal gives a good indication. The otitis externa always is confined to the membranous auditory canal. We find, therefore, after we have inserted an ear speculum through the narrowed part of the canal, that the bony auditory canal is of normal width and the tympanic membrane is unaltered. If,

on the other hand, we are dealing with an otitis media and a stenosis of the auditory canal caused by an affection of the antrum and the mastoid process, inspection of the bony auditory canal will show a sinking of the posterior upper wall, and the swelling of the external auditory canal is more pronounced in the bony part than in the membranous.

COURSE.

We distinguish three stages of the disease:

(1) The initial stage. This begins with the appearance of the ear symptoms and ends shortly afterwards, sometimes after a few hours, at the longest after three to four days, with perforation of the drum and purulent discharge in the external auditory canal. The violent pain and the fever usually last until the drum perforates and evacuation of the pus externally takes place.

(2) The second stage is characterized by suppuration. The patient is free from pain and the temperature is normal or slightly elevated. During the first few days there is a profuse purulent secretion. After about eight days the daily quantity of pus decreases spontaneously, and later on ceases. The secretion lasts for two to three weeks. During the last few days the secretion consists of almost pure mucus. The thicker and creamier the pus is and the more mucus the secretion contains and the less intense the secretion, the greater will be the danger that the secreted material will remain in the tympanic cavity or in the depths of the auditory canal, where it will either dry up or undergo putrefaction. The hearing will improve with the decrease of the suppuration.

(3) In the final stage, after the secretion has ceased, that is, one to two weeks later, the perforation will gradually close. Larger perforations require a longer time for healing; yet even with comparatively large perforations there is no danger of an opening remaining in the tympanic membrane, provided that the disease ran a normal course.

In all cases of otitis media suppurativa in individuals whose ears were normal up to the time of the disease, and where the disease appeared during the course of ordinary diseases, on the basis of chronic catarrhal processes in the nasopharynx, the prognosis is good for complete restoration of the ear, both

anatomically and physiologically, provided the patient was treated correctly and did not suffer from any intercurrent disease.

The cases of otitis media suppurativa on the basis of chronic catarrhal processes in the middle ear have a tendency to recover with permanent changes on the tympanic membrane and diminution of hearing.

The same is true of cases of otitis media suppurativa due to adenoid vegetations in the nasopharynx and catarrhal changes in the eustachian tube, but they will last longer.

Finally, we must deal with the danger of persistent changes in the middle ear due to an organization of the mucous secretion during the final stage of suppuration. These are the cases where the otitis media suppurativa gives rise to a chronic adhesive process. There is the danger of permanent changes on the tympanic membrane in all cases which require repeated paracenteses, as well as in all cases in which there is ulceration of the tympanic membrane at the perforation, due to the poor state of nutrition of the patient (cachexia, anemia, chlorosis, etc.). Finally, there is the danger of persistent changes on the tympanic membrane in cases of recurrent otitis media suppurativa, which is often observed as a special type of otitis during infancy. The earlier attacks will recover with complete restoration of the tympanic membrane, but the later relapses will lead to a cicatrization; and after the sixth to the eighth relapse, healing will take place with a persistent perforation.

This is due to the fact that during the course of the disease the lamina propria of the tympanic membrane will gradually become atrophic, especially in the region of the perforation.

We must not forget that in any severe case of otitis media the purulent inflammatory stage may be followed by a catarrhal purely inflammatory stage.

All cases of otitis media suppurativa acuta which appear during the course of acute infectious diseases, have a tendency to become chronic.

This is especially true of the otitis of scarlet fever and of measles during infancy, as well as of otitis media suppurativa appearing during the course of diphtheria and of typhoid. Tubercular otitis media suppurativa, also, shows a striking tendency to become chronic, as do the otitis media suppurativa

of the early stages of lues, and all cases of the disease appearing in otherwise weak, dyscratic or marasmic patients. A certain, though not great, tendency towards chronicity is to be found in cases of otitis media suppurativa of early infancy as well as of old age.

COMPLICATIONS.

In discussing the normal course of acute otitis media suppurativa, we saw that the inflammation, after the second week, is limited to the tympanic cavity and the antrum. We saw, also, that in normal cases the disease will involve the mastoid process and the eustachian tube only during its earlier stages. Complications are due to the fact that the mastoid process and the tube will take part in the inflammation during the later stages also, or that it extends beyond the limits of the anatomic middle ear and involves its vicinity. We distinguish therefore

(a) Intraaural complications.
(b) Extracranial complications.
(c) Endocranial complications.

Mastoiditis suppurativa is to be classed among the intraaural complications. Influenza otitis and typhoid otitis have a certain predisposition towards mastoiditis. Otitis caused by the streptomucosus, especially otitis caused by mucosus infection, is characterized by the rapid formation of an exudate which fills the entire middle ear, including the cells of the mastoid. Rapid softening of the bone is a characteristic feature of typhoid and influenza otitis. Mastoid processes with meningeal structure have a special tendency towards mastoiditis suppurativa. This leads to mastoid empyema through direct spreading of the pus. Diploetic mastoid processes are not so often attacked by the disease. In them, mastoid suppuration usually appears by metastasis. Rapid destruction of the bone is favored by rachitis during infancy, preceding constitutional diseases, as well as tuberculosis and lues. In tuberculosis, mastoiditis usually appears late and often has its origin in a tubercular periositis of the mastoid cortex. Retention of secretion in the tympanic cavity also favors the development of mastoiditis suppurativa from acute otitis media suppurativa. There is especially danger of mastoiditis in those cases in which the upper part of the tympanic cavity is primarily affected, and in which a small perforation in Schrapnell's membrane

provides the only drainage, also in cases of mesotympanic otitis, in which the perforation is to be found in the pars tensa of the tympanic membrane, but not in the level of the membrane, but rather in the mastoid protuberance.

Attention is called here to the fact that in the latter cases, the mastoid proturbance should be destroyed by paracentesis.

Pyorrhea of the tube is to be considered as a complication of acute otitis media. It occurs in cases of tubercular otitis media suppurativa, as well as in cases of simple otitis in which the patients have large adenoids.

There is great danger of affection of the middle ear and of labyrinthitis suppurativa in cases of fulminating otitis media suppurativa which appear during the course of scarlet fever, measles, diphtheria and typhoid. Metastatic infectious neurolabyrinthitis may be caused by mumps or influenza.

Cases of hemorrhagic labyrinthitis are sometimes to be found in cases of otitis media suppurativa with peracute leukemia. The temporal relation between the otitis and the labyrinthitis varies greatly. If the two appear simultaneously, we have to deal with a panotitis. Usually, however, the purulent labyrinthitis is preceded by the otitis media suppurativa. In rare cases of meningitis cerebrospinalis, the labyrinth suppuration may be followed by the otitis. In the acute stages of tubercular otitis media, a labyrinth suppuration is not to be found.

Among the extracranial complications, special attention must be called to the burrowing abscesses. These cases also depend largely on the character of the infection, for in a comparatively large number of extracranial complications and burrowing abscesses, the streptococcus mucosus can be found.

The abscesses appear more especially in cases of pneumatic mastoid processes, in which the corticalis of the pneumatic mastoid is thin. In infancy, the formation of subperiosteal abscesses takes place with a striking rapidity. Their development is especially favored by rickets and scrofulosis in the patient. Sinking abscesses burrowing towards the external surface of the base of the skull are found especially in tubercular patients.

Among the endocranial complications of acute otitis media, we must give first place, on account of their frequency, to pachymeningitis and extradural abscess. They are found more

frequently in the posterior than in the middle cranial fossa. If the extradural abscess is in the region of the sinus, we call it a perisinus abscess.

In the second place, we must mention sinus phlebitis, sinus thrombosis, pyemia, toxemia, and bacteremia.

About 50 per cent of all cases of inflammatory sinus phlebitis may be traced etiologically to an acute otitis media suppurativa. In the first year of life, however, sinus phlebitis complicating otitis media is rather rare. It is quite frequently found between the ages of ten and twenty, when it decreases in frequency but increases in danger as a complication.

In the first years of life we rather frequently meet with pachyleptomeningitis suppurativa in acute otitis media. In adults it is not so frequently a complication. Three types must be noted:

(1) Meningitis purulenta cum otitide media acuta, in which the meninges are affected simultaneously with the middle ear.

(2) Meningitis tuberculosa, of which there is always the danger in tubercular acute otitis media suppurativa.

(3) Meningitis secondary to acute otitis media suppurativa in older people, where the meningitis suppurativa appears acutely after an apparently favorable course of the acute otitis media. It usually ends fatally, sometimes after the primary disease has apparently gotten well.

In general we may say that there is greater danger of a meningitis in otitis media of the epitympanal type. In these cases the cavum epitympanicum is the primarily or exclusively affected part of the ear. The drainage of epitympanal otitis media is always difficult, and we are confronted with the special danger of a retention of secretion in the middle ear which greatly favors the extension of the purulent process to the meninges. In such a case, the disease usually extends into the middle cranial fossa, and in this connection the anatomic construction of the tegmen tympanicum is of importance. If it is either thin or dehiscent, the purulent process may make its way more directly and rapidly from the middle ear to the meninges than if the upper tympanic cavity were shut off from the dura by a compact bony covering. In cases of thin or dehiscent tegmen tympanicum, we will sometimes find intrameningeal abscesses of the middle cranial fossa during the course of the acute otitis media suppurativa. Purulent brain abscesses are very rare in acute otitis media suppurativa. Most cases of this

kind in the literature will not stand criticism. Usually, it is a case of chronic otitis media suppurativa with an acute exacerbation.

If the acute otitis media is not treated, there is the danger of pus retention with its consequences. The stagnant pus becomes putrid and fetid. There is eczema of the auditory canal and inflammatory swelling of its lining, with granulations at the perforation, which may become occluded. The pus in the tympanic cavity is forced into the mastoid process. There is no doubt that some cases of purulent mastoiditis are caused mechanically by the stagnation of pus in the cavities of the middle ear. Moreover, the pus may exert considerable pressure on the walls of the tympanic cavity. The head feels heavy, there is pain in the temporal region, the tegmen tympani becomes inflamed, and finally extradural abscess and meningitis occur.

In other cases, the pressure upon the lateral wall of the labyrinth becomes very great, and we observe spontaneous nystagmus and vertigo.

In neglected cases of otitis media suppurativa, the hearing will always be greatly diminished, and the danger increases with the duration of the suppuration, since the hearing will remain diminished after the disease gets well. If a neglected case recovers spontaneously—and we cannot deny the possibility of spontaneous recovery of an acute otitis media—the stage of suppuration will frequently be followed by a long catarrhal stage. In consequence of the organization of the exudate, the otitis media suppurativa may be followed by the formation of bands of connective tissue in the middle ear, i. e., a chronic adhesive process, which in turn results in a degeneration of the organ of Corti and labyrinthine deafness. It may be briefly stated that complications of acute otitis media suppurativa are increased by neglect of the disease.

It has already been stated that a permanent pyorrhea may follow an incorrectly treated tubal suppuration.

TREATMENT.

In the first stage, we content ourselves with purely symptomatic local treatment.

Violent earache is sometimes considerably diminished by instillations of a 3 to 5 per cent carbolic glycerin solution or an

adrenalin solution. If the auditory canal is wide, so there is no fear of a stenosis from epithelial maceration, we may instil a warm (40° C.) solution of aluminium acetate. Instillations of solutions of cocain, novocain and alypin have a good, though only temporary, effect. We use a tampon dipped in a 20 per cent solution, which is inserted as far as the tympanic membrane. The local application of cold (ice bag, etc.) is recommended during the first stage of the inflammation, while hemagogues are usually of no help. Rest in bed and a good evacuation of the bowels are ordered. Alcohol, strong coffee or tea are forbidden.

If the pain is very severe and is accompanied by fever, it will soon be seen that all anodynes are of no avail. In such cases it is advisable not to wait for spontaneous perforation, but to make an immediate paracentesis. A lance pointed knife mounted in a Politzer handle is used. The tympanic membrane is incised from below upwards, making the opening back of the umbo, and as long as possible.

If, on the other hand, circumscribed yellowish discoloration in the tympanic membrane indicates the imminence of spontaneous perforation, it will be necessary to make the paracentesis at that place, or at least to include it in the incision, otherwise it will be a useless paracentesis.

Sometimes it may be difficult to decide whether to make the incision or to wait.

If conservative treatment fails to give relief, i. e., if the pain and fever continues, paracentesis is indicated. As to the time to perform it, we must remember not to hesitate. It is better in doubtful cases to perform it than not. There is no danger so long as the operator observes all the aseptic precautions possible in the ear. The course of an uncomplicated inflammation of the middle ear may even be shortened by the fact that the serosanguinous exudate is evacuated externally through the opening, making its absorption unnecessary. Only in cases where aseptic precautions were not observed, do we meet with the danger that the uncomplicated inflammation may be converted into an infected purulent one through infection caused by the paracentesis. On the other hand, the omission of the paracentesis at the proper time, may cause an irretrievable injury, complications dangerous to life, and even death.

Preparations for performing the paracentesis.—The auricle

is cleansed with a tampon dipped in benzene, and the head is then covered with a fenestrated sterile compress, through the opening of which the auricle is drawn. The external auditory meatus is washed with a 1 per cent solution of hydrogen peroxid. For anesthetizing, instillations of novocain-adrenalin solution is recommended. This is prepared by heating 1 cc. of a 20 per cent solution of novocain or alypin to 40° C. and adding shortly before using it five drops of an ordinary solution of adrenalin, heated. The solution should be left in the meatus ten to fifteen minutes. The ear clinic of Freiburg recommends the infiltration anesthesia of the drum by injecting a Schleich-adrenalin mixture into the tympanic cavity (Tiefenthal, Bernd).

Instillations of 5 to 10 per cent aqueous solutions of cocain, with an addition of five drops of adrenalin 1-1000, have also been recommended. Eucain (8 per cent aqueous solution) is innocuous and can be sterilized. Gray's solution is also worth mentioning (cocain. muriat. 0.5, ol. anilini, alcohol. absol., aa 5.0). Ten drops heated to 40° C. are left in the auditory canal about five minutes. Haug's solution is useful (cocain. muriat. 1.5 to 3.0, glycerin 10.0, alcohol. absol. 10.0).

The operation should be performed under aseptic precautions, since otherwise there is the danger of causing a secondary infection. If the paracentesis has been performed at the proper time, immediately afterwards there is an evacuation of a serosanguinous or sanguinopurulent exudate through the opening. If, on the other hand, pure pus is evacuated, it is a sign that the paracentesis was performed too late, that the inflammatory exudate has been subject to purulent degeneration to such a degree that all the cavities of the middle ear are filled with pus (acute empyema of the middle ear). Before performing the paracentesis, strips of gauze should be dipped into a warm solution of aluminium acetate, and these should be introduced into the meatus immediately after the operation, without any previous cleansing of the canal. A moist dressing is applied to the ear, and the patient should be put to bed. In cases running a favorable course, a profuse discharge of pus will be seen for the next few days. Within three to five days, the temperature will drop to normal, and there will be no pain a few hours after the operation.

If the perforation is not within the level of the tympanic membrane, but rather at the top of a mastoid protuberance,

there will sometimes be an agglutination of the opening and symptoms of retention, in consequence of purulent stagnation in the mastoid process. In such cases the mastoid protuberance should be well incised with the paracentesis needle.

With the purulent secretion the otitis media suppurativa has entered into the second stage, and it is now necessary to take care of the discharge of pus and to prevent any retention or secretory stagnation. This is accomplished in different ways. Good success is obtained by moist dressings of acetic acid or 2 per cent alsol, renewed every day during the first few days. A moist strip of gauze is inserted into the external auditory meatus for the purpose of drainage, and after twenty-four hours we usually find the entire dressing saturated with pus. Later on, gauze strips 4 to 6 cm. long and 2 cm. wide are introduced. We may apply simple absorbent gauze dressings, or gauze impregnated with antiseptics (xeroform, vioform, dermatol, aristol, or ectogenous gauze). The insertion and removal of these gauze strips may be made by the nurse, the instrument, a blunt straight or angular forceps, being first sterilized by washing in benzene or by boiling. The dressing must be changed hourly or every other hour in case of profuse discharge, or two or three times daily if the discharge is slight. The point is that the strips should remain in the ear only so long as they absorb the secretion. If it is entirely saturated with pus, it must be removed before fresh pus collects behind it. We recognize that the dressing is being changed sufficiently often whenever there is little or no pus left in the external auditory meatus when the strip of gauze is removed, and whenever the patient is free from pain and the canal is open. Syringing is necessary only when the secretion is thick and ropy, and should be made with water at a temperature of 38° to 40° C. Insufflation of antiseptic powders is entirely unnecessary when the discharge is copious.

This method of treatment has the advantage that the physician is informed as to the condition of the suppuration, even if he doesn't see the patient daily. The latter will observe the decrease in the suppuration when the strips are not entirely saturated or are dry.

In a disease running a normal course, at the end of the first week of suppuration a viscid mucous secretion is found, which very soon thereafter becomes stringy. Therefore, simple re-

moval of the gauze strips will be insufficient, since the semi-solid pus has the tendency to remain in the tympanic cavity or in the external auditory canal. It is now advisable to use hydrogen peroxid (3 to 6 per cent hydrogen peroxid, or perhydrol 0.5, aquæ destil. 30.0), which should be heated to 40° C. and dropped into the ear two or three times daily. The pus is removed from the tympanic cavity three or four times a week by politzeration, and subsequent cleansing of the external meatus is done with cotton pledgets moistened with a 5 per cent aqueous solution of perhydrol. The hearing is improved by the politzeration, though it will become temporarily worse when the cavities again become full of secretion. Cautious aspiration by means of a Siegle's speculum or a suction bell will sometimes be beneficial.

Special care must be taken in connection with the external auditory meatus, since an eczema or an inflammation of it would increase the purulent stagnation in the middle ear. In the later stages of the disease, the ear must be inflated daily, until the hearing is permanently good, and the lateral surface of the tympanic membrane is pale and dry.

When the suppuration has ceased, the perforation will usually close in a few days. The first sign of the healing of the perforation is that the edges become white. This color is due to the young epidermis.

The opening closes by gradual diminution. The growth of the new epidermis takes its course from the center of the tympanic membrane towards the periphery. All local treatment at this stage is contraindicated (Politzer). The closure of the perforation is spontaneous, and the tympanic membrane and hearing become normal. After an otitis media of longer duration, a scar or intermediate calcareous deposit may develop in the drum. It is advisable to keep the external auditory canal closed by a small plug of cotton for some weeks after the cure of the otitis media suppurativa.

In order to prevent repeated attacks of otitis media and also catarrhal relapses, it is necessary to treat the nasopharynx, to provide a free passageway through the posterior nares, to treat the epipharynx, and to restore normal conditions at the pharyngeal tubal orifices.

As to the treatment of otitis in nursing infants, it differs very little from that of later years. Early paracentesis is im-

portant, as otherwise we may be surprised by a sudden onset of cerebral symptoms (meningism), or even by a purulent meningitis. An important point is the very careful treatment of the external auditory meatus, the cutaneous covering of which must be greased repeatedly. Free drainage must be provided for by thorough removal of the secretion and the macerated epithelial substance, which collects in the form of small scales. If the canal is constricted or obstructed by eczematous swelling, inflammations, etc., the treatment of the otitis becomes more difficult, and there may be a retention of secretion with the most serious consequences. Repeated paracentesis is recommended in cases which are very prolonged. Granulations should be removed, for if allowed to remain the course of the disease is more prolonged, and it may even become chronic on account of proliferation of the granulation. After the patient has recovered, it is often necessary to remove the hypertrophied tonsillar or adenoid growths, in order to avoid repeated inflammations.

VI.

PNEUMOCOCCUS INFECTIONS OF THE NOSE AND THROAT.

By Clement F. Theisen, M. D.,

Albany.

During the past fall and the present winter, some rather unusual cases of pneumococcus infections of the nose and throat have occurred in the writer's practice. Some of the cases were similar in many of their clinical aspects to the cases occurring in the epidemics reported in Boston, New York and other places, last winter. The name "septic sore throat" was given to some of the cases reported last year.

Most of the cases observed by the writer ran the course of severe infectious conditions, with marked constitutional disturbances and involvement of the kidneys. A more or less severe kidney disturbance is not very uncommon in throat infections, such as peritonsillar abscess, follicular tonsillitis and other inflammatory conditions.

The following cases are characteristic of the type seen by the writer this winter:

Case 1.—Child, aged seven months, admitted to the Child's Hospital in December, 1912, for an apparently severe cold. The child had a rather severe cough and a profuse nasal discharge of a stringy character. No pus could be detected in the discharge. The temperature was 102° F. on admission to the hospital, and almost immediately great difficulty in breathing developed. On account of the character of the nasal discharge, cultures were taken from the nose and throat.

On examination of the throat, the entire pharynx, including the tonsillar region, soft palate and uvula, was found to be extremely edematous, and the mucous membrane was covered with the same stringy mucus. Because of the great edema, a

satisfactory view of the larynx was not obtained. The glands on either side of the angle of the jaw were much swollen.

Two cultures from the throat were negative for Klebs-Loeffler bacilli, but contained pneumococci, and a swab taken from the nose alone, gave a pure culture of pneumococci. The difficulty in breathing for two nights was so extreme that we thought a tracheotomy would be necessary. There was undoubtedly a good deal of edema in the larynx also. That was six weeks ago, and there has been no recurrence of the difficult breathing. The edema of the pharynx gradually subsided under the use of a cold antiseptic spray. At no time was there any evidence of a collection of pus in the throat. The child is still in the hospital, and while the nasal discharge has stopped, there is still some fever. The blood count did not show any marked leucocytosis.

The child received one grain of urotropin in each ounce of fluid, for several weeks. Whether this modified the infection, I do not know. Reports as to the value of urotropin in air passage infections are contradictory, but it certainly seems to do good in some cases. The writer has never seen hematuria follow the administration of urotropin, although cases have been reported after only thirty or forty grains.

Case 2.—The next case is that of Mrs. H., aged 66 years, who said that she never had a sore throat until the one for which the writer was consulted developed. For years, from time to time, I have had occasion to treat her for chronic catarrhal otitis media. She has small submerged tonsils which had never given her trouble before. The attack started with a sharp chill and headache. There was severe dysphagia from the beginning of the attack.

Ordinary remedies were used for twenty-four hours, and when the writer was sent for the patient was sitting in a chair with labored stertorous breathing.

The throat presented almost the same appearance as in the other cases, i. e., great edema of the tonsils, soft palate, uvula; in fact, the throat looked like an inflated bladder.

Calomel was given at once, and after incision through the edematous parts, a culture was taken which showed a mixed pneumococcus and staphylococcus infection with the pneumococci greatly predominating. Albumin and granular and hyalin

casts were found in the urine. Blood count showed a leucocytosis of 12,000. Ice packs were used around the neck, with ice in the mouth. An iced spray was used at short intervals, and urotropin was given, about forty grains daily being used, each tablet being dissolved in a large glass of water. Diet was limited to milk.

The edema of the pharynx persisted for a week, in spite of repeated incisions and local treatment. At no time was there any pus found. The throat gradually cleared up, but albumin persisted in the urine for some time. It is interesting that in this case the anterior surface of the epiglottis was also decidedly edematous, like the condition described by Michel[1] in 1878, to which he gave the name "angina epiglottidea anterior."

The writer in July, 1900,[2] also reported a series of cases in which the edema and inflammation of the anterior surface of the epiglottis occurred as a primary infection, without involvement of the pharynx.

In this series of cases, which also ran the course of severe acute infectious conditions, cultures showed mixed pneumococcus, staphylococcus and streptococcus infections, with the pneumococcus predominating.

Case 3.—The third case which the writer would like to report was similar to the last one, occurring in a man, aged 45 years, who had been subject to sore throat at times. This attack also started with a chill, headache and a sharp elevation of temperature (104° F.). The throat presented almost identically the same appearance as in the second case, except that the epiglottis was not involved. The great edema again persisted for a week, in spite of scarification and energetic local treatment. Albumin and casts were present in the urine, the spleen was enlarged, and in fact all the symptoms of a severe infection were present. Urotropin in large doses and in large quantities of water was given, and appeared to cut the attack short. Cultures in this case again showed almost a pure pneumococcus infection. The infection was so severe that Mulford's antipneumococcic serum was used, but without apparent effect. This case ran the same course as the second one, the throat slowly clearing up. Fairly high temperatures persisted for two weeks, and there was great prostration. No pus could be found in the throat at any time.

Cases 4 and 5.—Two cases, both young adults, were seen, with great inflammation and edema of the anterior surface of the epiglottis. The pharynx in both cases, with the exception of a slight redness, was normal. In both cases there was considerable difficulty in breathing, and great pain in swallowing; in fact, for several days they were practically unable to swallow. Cultures from both throats, after incisions, showed pneumococcus infections with a few other organisms.

These cases are of considerable interest, because they prove, contrary to Semon's[3] opinion, who claimed "that many of the forms of acute septic inflammation of the throat, that have been classified in the past as separate conditions, have a pathologic identity," that an acute edema of the anterior surface of the epiglottis may occur as a primary condition.

An interesting case of this kind has been reported by Crisp.[4] In this case, that of a boy eight years old, death occurred suddenly, apparently by suffocation, after he had been ill three or four days. The autopsy revealed a swelling of the epiglottis with no lesion in any other part.

Another interesting case has been reported by Fredet.[5] In this case, that of a young man of 20 years, great difficulty in breathing came on suddenly after a debauch. Suffocation rapidly ensued, and at the autopsy great edema of the epiglottis was found. When this was incised a large amount of pus was evacuated.

The fact that in the two cases of this kind, and in the three other cases reported by the writer in his paper, in all of which there was great edema associated with the symptoms of an acute infectious condition, and that they were all practically pure pneumococcus infections, is of some interest.

It is known that the pneumococcus, when some of the lower animals are inoculated with it, will sometimes produce local edema.

Welch,[6] in his interesting article on the "Micrococcus Lanceolatus," has brought this out. He quotes Foa, who distinguished a special variety of this organism. His pneumococcus he called diplococcus lanceolatus of the edematogenic variety. This edematogenic variety of the pneumococcus often produces, after subcutaneous inoculation into the subcutaneous tissue of some of the lower animals, widely spreading or local edema. Welch has also obtained cultures which when injected sub-

cutaneously produced uniformly extensive local subcutaneous edema.

Another point of interest in this series of cases, is the absence of suppuration. In none of the cases was pus found when incisions were made.

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VII.

TWO CASES OF AIR EMBOLUS FOLLOWING EXPLORATORY PUNCTURE OF THE ANTRUM OF HIGHMORE.

By H. M. Bowen, M. D.,

Toronto.

Exploratory puncture of the antrum of Highmore is looked upon as a very simple and harmless procedure, and is now practiced very extensively; but that serious and even fatal results may ensue, is shown by the following cases:

Case 1, March 5, 1910.—Patient, female, applied at the dispensary for treatment, and after the preliminary examination it was decided to wash out the antrum of Highmore. An application of a 20 per cent solution of cocain was made to the inferior meati and about fifteen minutes given for cocainization.

The puncture was made with some difficulty, owing to the unusual thickness of the bone. After inserting the needle, it was found impossible to force the air through with the syringe, so the needle was withdrawn slightly for a second attempt, which was also unsuccessful. During the third attempt the patient, without the slightest warning, became rigid and cyanotic, resembling very much an attack of epilepsy. A moment or so later there were convulsive movements of the extremities, frothing at the mouth, and stertorous breathing.

She was placed in the recumbent position, with the expectation that consciousness would return in the course of a few moments, but, on the contrary, she remained in this condition for seventy-two hours, and did not become rational until the evening of the fifth day.

As she did not respond to the ordinary stimulants, she was admitted to the hospital for observation. At eleven o'clock that night the temperature was 101 2-5°, pulse 75, and respirations 20. The temperature remained around 100° until the

third day. She vomited the first evening, and again the evening of the third day. There was profuse perspiration, especially at night, and muscular twitching almost continuously. Occasionally she became quite violent and required the use of a restraining sheet. During the fourth day there was involuntary micturition, and during the lucid period that evening she complained of pain in the abdomen. During the fourth day a partial paralysis of the left arm was apparent. She was able to sit up in bed on the fifth day and would converse when spoken to. She was discharged on the seventh day.

Case 2, July 25, 1911.—Patient, male, age 24 years, over medium height, muscular physique. His application for the police force of New York had not been considered, owing to defective nasal breathing.

Examination revealed a marked deviation of the septum for the relief of which he submitted to a submucous resection.

One month later he again applied at the dispensary, complaining of nasal discharge and alternating nasal obstruction.

The inferior turbinate on each side appeared largely swollen and of a dull blue color. The outer wall of the middle meatus had a peculiar dull gray, snail track-like appearance, presumably due to the flow of mucopus down the middle meatus, and under the assumption that one of the accessory sinuses was infected, it was decided to begin the search for the pus by puncturing the antrum.

Accordingly, the inferior meati were cocainized with a 20 per cent solution. The right side was washed out with a negative result. The left side was punctured in the same manner, but as the air was forced in with the syringe the patient became rigid, eyes half closed, eyeballs rolled backward, then relaxation, profound cyanosis, and slight muscular twitching. He was placed in the recumbent position, and outside of a few gasping respirations, at the rate of about three per minute, there was little evidence of life.

He was immediately given a hypodermic of strychnin, adrenalin, minims xv, several hypodermics of whiskey, artificial respiration being kept up all the while.

At the end of about twenty minutes the cyanosis cleared somewhat and it was thought that the danger was over, but in a few minutes it deepened again, and death occurred approximately one hour after the puncture.

A postmortem was performed the same evening. When the calvarium was removed, the sinuses of the dura were revealed, greatly distended, but there were no apparent changes around or in the substance of the medulla. Examination of the abdominal contents revealed old adhesions around the appendix and gall bladder.

When the sternum was removed, the pericardium immediately bulged forward into the opening. An incision into the pulmonary artery was followed by a sharp hissing sound of escaping air and a collapse of the distended heart.

The cause of death was quite apparent, but the condition of the antrum was most interesting of all. The puncture in the antrum wall was easily found, and when the anterior wall was removed, the mucous membrane of the roof, except for a small part, as well as the outer and inner walls, was found detached.

The mucous membrane covering the roof was hanging about a quarter of an inch below, and it showed a small puncture mark. The bone of the roof and posterior wall did not show any puncture mark, consequently the needle did not penetrate beyond the antrum.

The inner wall of the antrum was removed and the ethmoid was cleared out up as far as the sphenoid, but no large or misplaced vessels were found. There was not the slightest trace of hemorrhage in the antrum.

Taking everything into consideration, the only explanation which could account for the passage of air into the general circulation, would be that the needle penetrated the mucous membrane of the roof and the air was forced between the mucous membrane and the bone, opening up numerous capillaries.

Up until this time the technic employed was to blow air through the antrum before washing out. In this case there was no unusual force employed in blowing the air through, consequently it will readily be seen that it is a very dangerous practice. It does not indicate anything further or accomplish anything in preference to the solution.

In using the solution first, providing the needle did puncture the mucous membrane of the outer wall, no harm would be done by the injection of a small amount of sterile saline solution into the general circulation.

VIII.

NASAL HEMORRHAGE FOLLOWING TURBINECTOMY IN A HEMOPHILIAC TREATED BY THE INJECTION OF HUMAN BLOOD SERUM.

By Linn Emerson, M. D.,

Orange.

The use of human or other blood serums in cases of hemophilia is proving to be a measure of value. In the journal of the American Medical Association of October 26, 1912, four cases are reported by different authors, in which the measure was apparently successful in three.

Dr. Clement F. Theisen has also reported several cases. Dr. J. H. Bradshaw of Orange reported to the Orange Memorial Hospital Medical Society in September a case of hemophilia in the newborn, saved by the use of human serum, and described the apparatus used by Welch and himself to secure the blood for the serum.

Mr. J. C., aged 31 years, a vigorous, healthy married man, consulted me on September 28, 1912, for chronic catarrhal deafness. To improve hearing and facilitate the passage of the eustachian catheter, I removed the major portion of the left inferior turbinate on the morning of October 6th. As the bleeding was rather free and continued to ooze even after packing with Simpson tampons of Bernays' sponge, I advised him to remain in my office during the afternoon, as I was called out of town to perform several adenoid operations.

I had just begun my second adenoid operation when I received a call from his family physician, who was with him in my office, and who seemed to think the case needed my immediate attention. As I was more than twenty miles distant and had four other adenoid operations scheduled, all of whom had been starved and prepared, I could not see the feasibility of an immediate return, but advised him to either pack the

patient himself or get some other rhinologist. This he was unable to do, it being late Sunday afternoon and no other specialist being available.

I suggested serum injection, and the patient was given 2000 units of diphtheria antitoxin. On my return at 8:20 p. m., this was repeated, and the bleeding being persistent, I removed the tampons and packed tightly with a cotton tampon behind and gauze saturated with adrenalin in front. Even after this packing the blood oozed steadily from the front, and one or two ounces were lost during the night.

A special nurse was secured and the patient remained in bed in my offices, and I also slept there that night. About noon the following day the hemorrhage being nearly stopped, he was taken to the Orange Memorial Hospital in an ambulance.

The slightest movement or lying down would cause the oozing to start afresh from the front, so the patient sat up in bed for more than a week after the operation.

On the fourth day after he was plugged, I removed the posterior plug without mishap, but he was so fearful that the removal of the gauze would cause renewed bleeding, that he was permitted to pull it out himself and cut it off an inch at a time. Ten days elapsed before the gauze all came away, and the day following he was allowed to go home. Despite the fact that he remained very quiet most of the time in bed, bleeding recurred on the 20th, two weeks from the day of the operation, and after three days of milder methods of treatment I was obliged to plug him tightly again at 3 a. m. on the 23d. On this same day the patient's brother was brought from a distance, and I called Dr. Bradshaw to get blood from him for the serum. The apparatus did not work satisfactorily, and most of the blood was collected by spontaneous flow from the median basilic vein.

The serum was separated by clotting and gravity, and about seven ounces were secured. The first two days 20 cc. were injected subcutaneously, three times a day, and the effect immediately following the injection was marked. After two days the frequency of the injection was lessened, and when reduced to once daily the bleeding recurred, but to a much lesser degree.

The patient's physical and mental condition were becoming alarming, so it was decided to secure more serum. A healthy

man was secured, and on October 30th I bled him from the median basilic vein, collecting the blood in a wide-mouthed dressing jar; when the blood spurted, it was deflected into the jar by the cover held by a nurse. More than a quart of blood was secured, and about nine ounces of serum was obtained. This was used as before, and after using 20 cc. three times a day for three days, the packing was finally completely removed on November 2d. The injection was continued once daily for nearly a week after this.

The patient has very slowly and gradually regained his health and strength, but has been very apprehensive of a return of the hemorrhage. As late as February 1st he came to me, greatly worried because the mucus from his nostril was occasionally slightly tinged with blood.

His early history, secured by careful questioning after the operation, is interesting. He once had a tooth extracted and bled for three days afterward, but does not seem to have lost any very great amount of blood.

While a college student his leg was bruised while playing football, and a large swelling developed which "laid him up" for nearly a year. Several physicians were much puzzled at this condition, which was no doubt an intramuscular hemorrhage.

He had seven and one-half grains of calcium chlorid three times a day and gelatin dessert with his meals for a week before operation. This was also kept up for a number of weeks during his convalescence.

It is interesting to note that about a pint of serum, more than half of which came from a donor unrelated by blood, gave rise to no unfavorable symptoms of any sort.

IX.

ON THE FUNCTION OF THE TONSILS.*

By Louis M. Freedman, M. D.,

Boston.

In the consideration of the function of the tonsils, we are entering one of the medical fields of controversy which has lasted a long time. It has been difficult to ascribe a function to a tissue which, after its removal from the body, has not apparently been missed. The main cause of this controversy has been whether we should interpret tonsillar conditions in a pathologic, injurious light, or in a benignant, salutary light. There have been many theories regarding tonsillar function, none of which have in any way been definitely proven true. Yet, we are more definitely certain that the activity of the tonsils as such are of temporary use at least. We are at least certain enough about a possible function to make us hesitate in the use of radical tonsil surgery.

To better understand the possible functions, we must briefly consider, in outline at least, the anatomy of the tonsils. The tonsils are composed of lymphatic cells, collected in groups called follicles. These follicles are separated from one another by areas of connective tissue. These masses so formed are held together externally by a fibrous capsule which binds the tonsil to the tonsillar sinus—the triangular space between the anterior and posterior pillars of the soft palate. The internal surface of the tonsil, facing the oral cavity, is lined by a flat epithelium. The epithelium of the mouth and nasopharynx is generally ciliated, and directs particularly toward the tonsils. The surface of the tonsil is irregular and indented with crypts, ten to twenty in number, which have opening into them the ducts of the muciparous glands situated in the

*Read at the fall clinical meeting of the Staff of Mt. Sinai Hospital, Boston, November, 1912.

stroma of the tonsil. Surrounding each follicle is a close plexus of lymphatic vessels. From these plexuses the lymphatic vessels pass to the submaxillary lymph glands below the angle of the jaw. From the submaxillary lymph glands the lymph goes to the deep cervical glands. The main blood supply of the tonsil is the tonsillar artery, a branch of the facial artery, although many other vessels contribute in part to the arterial supply of the tonsil and its surrounding tissue.

In development the tonsil is not quite mature at birth; but at the end of the first year it is fully formed and active. If it functions at all in any special way, it does so in the early years of life, most likely before the age of seven. After that the tendency is for it to atrophy. A tonsil remaining large after that period is generally not found to be normal, at least in its activity. Thus, in the tonsil we have a lymphoid structure with a short period of usefulness.

In the study of the physiology of the tonsil, many hypotheses have been advanced concerning its function; some of these are not rational, others are very suggestive. Flak holds that the tonsils are residual embryonic remains. In favor of this is urged the fact that in intrauterine life the tonsil becomes markedly developed, and in early life it tends to disappear. Against this is the fact that the tonsils have been traced in all the animal forms from reptile to man, and it is most highly developed in man. Another old theory was that the tonsils had some necessary and obscure relationship to the organs of generation, and removal of the tonsils would cause sterility. The latter part of this statement has been absolutely proved untrue. Whatever possible relationship there exists between these tissues, it is certainly not a necessary one.

The theory that the tonsil is a blood forming organ in early life, and that after the tonsil has passed its active period this function goes over to the bone marrow, has had many supporters. Harrison Allen holds that the tonsils are a tissue of the type we see in the thymus, lymph glands, and the bone marrow. When the thymus retrogrades, the tonsils develop and take up its function; these in turn retrograde and in turn the bone marrow takes up the function at the full growth. This is not rational; for, long before the thymus has reached such a stage, the tonsils and lymph glands are not only developed but hyper-

trophied. Further, to take this viewpoint would mean to consider the hypertrophied tonsil most active at a time when it should be retrograding; yet under such conditions it does not alter or lessen the activity of the bone marrow. Further, even if it were possible that the thymus, tonsils and bone marrow might be active vicariously at the unstated periods, it is still anatomically unreasonable; for, structurally they are each different. The thymus and bone marrow each lack germinal centers; the bone marrow lacks follicles; and in the lymph glands are lacking the various stages of development of the red blood corpuscles which we find in the thymus and the bone marrow. The anatomic differences lead to the conclusion that the tonsils and the lymph glands are the origin of the lymphocytes, and the bone marrow of the myelocytes. We have no evidence to show that the tonsils are of great significance in blood building. But the anatomic similarity between the tonsils and the lymph glands is so great, that one must admit that the tonsils are a part of the lymphatic structure and its system. Wood considers the tonsil a lymphocyte forming organ. According to him, the origin of the lymphocytes is not in the germinal centers, but rather the epithelium of the tonsillar crypts. He has seen all the transitional forms to leucocytes. In this belief he is apparently alone. The crypts are lined with a stratified epithelium which is not of mesoblastic origin. To agree with Wood is to overthrow all our views in relation to the mesoblastic origin of the leucocytes.

That the tonsil is a secreting organ many sought to prove. Some thought that it held a digestive ferment which converted starch to sugar. No secretion of such a nature has been obtained. Others thought that the tonsil secreted a mucus which facilitates deglutition by moistening the bolus of food. Likewise, this has never been demonstrated. Finally, an internal secretion such as the thyroid and adrenals have, has been sought for in the tonsils. Masini injected tonsillar extracts into animals, and thought he obtained a rise of blood pressure such as is seen with adrenal extracts. Scheier, in covering the same ground, got a notable fall in blood pressure. Pugnat, repeating these experiments, got neither rise nor fall in blood pressure. It is evident, therefore, that with the three possible conclusions obtained by three different men, that at least two of them prob-

ably had some error in technic that led them to the wrong result.

The late Dr. Nelson of the Harvard Medical School and myself undertook to make tonsillar extracts, for this purpose. We divided the work into two parts. First, we made extracts of the extracellular substances; second, we made extracts of the intracellular substances. The extracellular extract was made by chopping up the tonsils finely, and rubbing them up with sand mixed with .8 per cent normal saline in a mortar. This was allowed to stand for a few hours and then filtered. The filtrate was then injected into the jugular vein of a properly anesthetized rabbit, so placed that the recording apparatus for blood pressure and respiration would register the changes. The extract was injected in varying doses and at varying intervals, but no change of any sort was noted. By way of control, a small portion of the filtrate was treated with alcohol and a precipitate obtained, showing the probable presence of an albuminous substance, and that the filtrate was not absolutely inert. Further, as another control a 1-10,000 solution of adrenalin was injected into the rabbit in very small amount. This showed a tremendous rise in blood pressure. This showed quite definitely that the failure to get an effect upon the blood pressure with the extracellular extract was not due to the inertness of this extract, or to any difficulty with this particular animal, but rather because an aqueous or normal saline extract of the tonsil does not contain any blood pressure altering substances. The extract for intracellular substances was made by similarly cutting up the tonsillar tissue very finely; but this time, instead of the saline solution, a solution of cupric chlorid was used to dissolve away the cellular envelope. This was allowed to stand forty-eight hours and then filtered. The clear blue filtrate was then treated with H_2S gas to precipitate the sulphid and thus remove the copper salts from possible activity. The colorless filtrate resulting was then injected into a rabbit, under the same condition as before stated for the salt solution extract. The result was likewise negative. The same controls were used as with the salt solution extract, with the same result. In doing this work we found how easy it was to be led astray by a slight error in technic. For instance, the first extract we used with cupric chlorid, we did not free entirely of

its copper content. This resulted in causing a marked fall of blood pressure in the first part of the curve, which no longer occurred when the rest of the filtrate was freed of the copper content entirely. It would seem then from the foregoing, that no internal secretion which affects blood pressure is present. This certainly agrees with our clinical experience. For, with tonsillectomy being so widely practiced in America, almost to the exclusion of any other method among some very active operators, we have as yet not been able to discover that any necessary secretion has been lost; or, at least, if such a secretion has been removed, other organs continue to supply it in sufficient amount for the individual, so that no loss is felt.

We come now to the theories that give to the tonsils a protective character for the body, in contradistinction to those theories which have for their basis that the tonsil is a portal of entry for infection, and therefore a source of danger.

Ashhurst holds that the tonsil is a protective organ, but from an eliminative point of view. To support his view, he finds it significant that in diphtheria, scarlet fever, and other exanthemata, the tonsils are affected after other manifestations of the disease have been present days or hours. To further support his view, he cites the case of a physician who pricked his finger when opening a postscarlatinal abscess. The next day he attended another septic case, during which he noticed that that finger was uncomfortable. Following that, his finger became swollen, and his arm likewise, with a resulting lymphangitis. Then constitutional symptoms such as slight fever and malaise intervened. Two days after that the tonsil of the same side became inflamed, and later the tonsil of the opposite side also. But no cultures were taken. This he considers a sort of elimination. In the same way he explains the obscure relation between articular rheumatism and tonsillitis, and endocarditis. The position does not seem tenable. In the diphtherias and the exanthems it has been as frequently noted that the tonsil affection precedes the other symptoms as it follows them. Anyone who has treated many of these cases has had for a few days or hours, no more than the throat affection and temperature to go by for a diagnosis. This is certainly true of the diphtherias. In measles, the coryza and lacrimation are the first signs. So likewise we

more frequently see the tonsillitis anticipate the articular signs in rheumatism than follow them. Further, the case of the physician cited is open to a number of possible interpretations, of which the elimination theory is the least likely of all the solutions. With no culture taken there is not even the evidence definitely that the same organism was present in the arm as in the tonsils. Is it not more likely that the organisms already present in the tonsils received new stimulation for activity with the lessened general body resistance of the patient, when an active septic process was present in the arm? Ashhurst further holds that the tonsillar hypertrophy is not simply a local condition, but is rather evidence of a diathetic taint or condition of malnutrition. Removal of such hypertrophy would be a benefit to the general health by removing a mechanical obstruction to respiration; and secondly, it would improve the function of the tonsil, whose crypts and recesses are, in the process of hypertrophy, made deeper, and their normal outflow thereby impeded. The accumulations in these recesses are a source of danger to the entire organism. It is difficult to be contented with the supposition that tonsillar hypertrophy is evidence of a diathetic taint; for, if that be true, a tremendously large proportion of the children of America and Europe have that taint. Further, we see it as frequently in robust and well nourished children as in poorly developed types. It would seem that tonsillar hypertrophy might in fact be an evidence of beneficence, if we believe in the doctrine of the "survival of the fittest."

It has been a much mooted question whether the normal direction of the lymph flow in the tonsil is inward or outward; and on the answer to this question depends the protective character of the tonsil. We have seen that the Ashhurst view is that the protection is by a sort of elimination outward.

Stöhr discovered that through the epithelium of the tonsil there is a constant stream of leucocytes wandering forth. The leucocytes swarm about like patrols or watches, and fall upon the foreign particles, take them up and destroy them by phagocytosis. Brieger, however, showed that it was not the leucocytes which come forth, but rather the lymphocytes, and they are not supposed by the physiologists to be phago-

cytic in character. Further, he contends that the lymphocytes do not of themselves actively and independently change their position, but are passively transported from the tissue of the tonsils. This medium of transportation can be nothing other than the lymph fluid which fills the interstices even through the finest structure. This is not really emigration, according to him, but rather a streaming through, or "Durchströming," as he calls it. The protective quality is due, first, to the mechanical outflow of the lymph, which thereby hinders the entrance of foreign substances; and secondly, to the bactericidal effect of the lymph fluid, which is identical with the blood serum; thirdly, it is due to bactericidal substances set free in the decomposition of the lymphocytes after their exit from the tonsil. Stöhr does not agree with this. He says that the lymphocytes wander out through the epithelium, and the places where they come through may serve as entrance points for bacteria and foreign substances, and thus are a source of infection in the form of "physiologic wounds or spaces." From the Brieger viewpoint, the lymph fills out these spaces so that they are not a source of infection. It is to be admitted that the crypts are a great source for infection; for they are filled with collections of bacteria, detritus, and whatever foreign substances might enter from the throat. It is, however, difficult to believe with Stöhr, that any tissue of the body is formed primarily to be a source for infection.

Goodale experimented with the absorptive power of the tonsil for foreign particles. He injected into the crypts of hypertrophied tonsils, under moderate pressure, a watery solution of carmin in fine suspension, by means of a dull pointed cannula attached to a hypodermic syringe. The tonsils were removed at varying intervals following the injection. The first ones were removed twenty minutes after the injection, the next forty-five minutes after injection, the third an hour, etc., and the last ten days after the injection. The tonsils were imbedded and serial sections cut. The sections showed the carmin in the crypts, from which fine lines radiated up to the epithelium. Where the structure of the epithelium was loose, the particles entered deeper into the cellular layers. Some of the particles were close to the leucocytes, others were within the leucocytes. How deeply the carmin entered into

the tissue, depended upon how long after the injection the tonsil was excised, and the looseness of the epithelial layers. In no case were the particles found within the follicles. In the case where the carmin had been injected ten days before, there was very little carmin in the crypts; but most of the carmin was in the deeper interfollicular tissue, radiating in the direction of the deeper connective tissue layers. Each case showed bacteria in the crypts, but none in the tonsillar structure. In one case a solution of carmin in which a characteristic bacillus had developed was injected. Two days later the tonsil was removed and examined. The bacillus was found in great numbers in the crypts, free and in neutrophiles; yet none were found in the tonsillar tissue, although in the interfollicular spaces much carmin was found. Further, four cases of acute follicular tonsillitis were examined histologically for bacteria. Though many bacteria were found in the crypts and mucous membrane surface, none were found in the tonsillar tissue. Brieger objected to these results, on the ground that possibly undue pressure had been used in forcing the fluid into the crypts, so that it was not absorption but pressure that brought the carmin into the tonsillar tissue. But in this he was wrong; for the first tonsil, removed after twenty minutes, showed only the presence of carmin in the crypts, not the tonsillar tissue. Further, had pressure been the actual means of introduction into the tonsillar tissue, then the bacteria would also have been unavoidably forced along with the coloring matter. These experiments, therefore, show that the tonsils absorb foreign particles, but not bacteria; and that inflammations in tonsils are primarily set up, not by the bacteria, but rather by their toxins.

Schoeneman considers the tonsil a lymph gland, like the rest in the body, only it is submucous in position and has for its main function the drainage of the nasal mucous membrane. This he proves by painting the nasal mucous membrane of one side with coloring matter, which he later finds in the tonsil of that side. He likewise explains the tonsillitis which follows the socalled head colds; and so also the tonsillitis which follows operative measures on the nose. Tonsillar hypertrophy is to him comparable to simple chronic lymphadenitis.

Further, on the absorbent power of the tonsil, George Wright of Boston has shown a definite relationship to the teeth. He shows that the enlargement of the tonsils without necessary inflammation may occur, and usually does, at the correspondingly same periods as the four periods of molar dentition: at two years, six years, twelve years, and at seventeen years. Prophylactic treatment of the teeth at these periods will cause reduction of the tonsillar enlargement. In the development of the teeth at these periods, a dental sac, analogous to the placenta, is formed to carry all the materials necessary for the production of the enamel and dentine and their nutrition. "The final disposition of what remains of this organ after the teeth have erupted, together with the bone and connective tissue surrounding, also the roots of the temporary teeth are involved in lymphatic absorption. With the upbuilding of forty-eight teeth and the changes occurring in the jaws, there is likewise much waste tissue for disposal." Wright believes that the tonsils together with the lymph glands take care of these waste substances. So likewise, with decayed teeth, the tonsils are enlarged to absorb the waste and septic material. If the teeth are cleaned up, thus leaving less for the tonsil to absorb, the tonsillar enlargement does not occur or subsides.

Jonathan Wright finds, from an evolutionary point of view, something significant in the enlargement of the tonsils in civilized children in response to changes of temperature from the overheated house to the excessive cold out of doors. In the system there is a call made upon the vasomotor and cellular activities; in the response to this we get an ontogenetic and phylogenetic adaptation. This response is usually a congestion, and often an inflammation, resulting in the hypertrophy of the pharyngeal lymphoid tissue. Enlarged tonsils, as has already been previously noted, are as much if not more often present in robust as in frail children. It is also a fact that the submerged tonsil is more the tonsil of infection than the large free one. The relatively greater frequency of enlarged tonsils among the children of civilized man, apart from the barbarous races, goes hand in hand with their greater natural immunity against many infectious processes. It is a well-known fact that the American Indian has not been prone

to have large tonsils; and yet, the ordinary infectious diseases of childhood have a more serious effect upon him.

R. H. Good, and likewise Sheedy, think that the tonsil may in early life act so as to immunize the system against many diseases, the crypts acting like culture tubes in which various toxins are elaborated and introduced into the system in quantities sufficient for immunization. He carries it still further, by calling attention to the fact that children are more likely to have certain contagious diseases that adults rarely get—due to the immunity established thus in childhood. Because of the location of the tonsils, bacteria entering the oronasal cavity are directed toward the tonsillar crypts. The crypts are simply culture tubes or receptacles in which bacteria of every variety may be cultivated—the mucus derived from the glands outside of the tonsillar structure, acting as a culture medium. The bacteria multiply in the crypts, give off products (vaccines) which are taken into the tonsil by the lymph currents and thence into the entire system. When these products come in contact with the fixed tissue cells, antibodies, agglutinins, opsonins and so forth are produced. Thus are the leucocytes prepared to exert their phagocytic action. This process of leaving the bacteria in the crypts long enough to establish an immunity and then be carried off by the phagocytes produces a socalled tonsillar equilibrium.

With so many possible theories, a number of which bear the stamp of having at least a kernel of truth and plausibility, it is very difficult to properly come to any definite conclusion in regard to the functional properties of the tonsils. Yet some facts stand out quite definitely. It is evident that the tonsil is a lymphoid structure such as any lymph gland; it is connected with the lymph glands and channels, and has the advantage or disadvantage of being superficially placed. But it is apparent that its period of activity is only for the early years of life. It is very likely that the tonsils play some part in blood building, but that is not very important for the system, because it is not the only organ for this purpose, and also because it is apparently not the important part of its work. It is reasonable to believe, because of the leucocytes present and their noted phagocytic character, that they are protective during their period of normal activity. The ex-

periments of Goodale showed quite conclusively that this protective character against bacteria was quite definite. It seems fair to call the tonsils, as many have, "the sentinels of the body." When an infection enters through the tonsils, there is great likelihood that the tonsil attempts to overcome the invaders, and failing in the attempt, the next lymph glands in order of defense—the submaxillary—take up the battle; and if any of the invaders sift through further, the cervical lymph glands become the defenders. Yet it must be noted that in the usual tonsil infection it is not the bacteria but their toxins which have apparently set up the inflammation. Whether a process of immunization actually takes place, of course, is not definitely proven, but there is certainly much in favor of such a theory. However, if the tonsil is so diseased that its ability to cope with an infection is so far reduced that on very slight provocation it becomes inflamed, and too frequently in the battle between it and the invading bacteria it becomes the loser, so that constitutional symptoms intervene, it is then fair to presume that no longer is it a sentinel, but rather an obstacle to defense, and may then actually serve as a portal of entry for disease.

The tonsil is so often spoken of as a portal of entry for disease that many have come to the conclusion that this is its function. That, on the surface, is not rational. It is hard to believe that the particular function of any organ or tissue of the body should essentially be injurious. It is, however, reasonable to believe that through disease any organ may be perverted in its function and thus serve as a source of danger to the entire system. Lexer painted virulent cultures of staphylococcus, pneumococcus and streptococcus on the tonsils, and set up an inflammation in the tonsil and resultant constitutional symptoms; he did the same on the mucous membrane of the throat, with a resultant inflammation and constitutional symptoms. This makes it quite evident that the tonsils per se are not simply portals of infection; for, given the same conditions, the pharyngeal mucous membrane will also be a mode of entrance for infection. Menger demonstrated in cases of acute articular rheumatism following tonsillitis, bacteria under the epithelium of the tonsil mucous membrane; but he found them likewise in the mucous membrane of the

neighboring tissues, and in these tissues they were deeper and much further advanced from the surface to the capillaries.

The tonsil is supposed by many to be quite frequently the mode of entrance for many diseases, notable among them tuberculosis. Dieulafoy took the tonsils and adenoids of ninety-six individuals, cut out the centers of these tonsils and adenoids, and put them under the skin of guinea pigs. He claims to have obtained as a result of the tonsils, tubercular infection in 12 per cent of the animals; and as a result of the adenoids, tubercular infection in 20 per cent of the animals. A number of men have repeated these experiments and have failed to obtain any striking results, except from a negative point of view. A year ago the writer repeated these experiments. Tonsils and adenoids were selected from fifty children —some of whom were tubercular and others of whom had a tubercular family history; these tonsils and adenoids were put under the skin of guinea pigs, as was done in Dieulafoy's cases. Of these, six died of streptococcus infection within a week, three died of pneumonia, only one showed any tubercular lesion, and the rest were prospering very notably when last seen, at the end of three months.

It would seem from the foregoing that the tonsil under ordinary conditions is not a portal of infection any more than any other tissue of the body, but rather a part of the protective mechanism of the system. It is also true, however, that when in a diseased condition, like any other diseased tissue of the body, it may become a mode of entrance for infection. This, however, is dependent upon other factors also. These factors are, first, the particular resistance of the individual; and second, upon the degree of virulence and pathogenicity of the bacteria concerned in each infection. Further, the tonsil, from its strategic position in the throat, is in the pathway of infection coming from many sources, and is perhaps in that way more open to infection than most of the body tissues.

In conclusion, it is fair to assume that the tonsil has a function, but probably only in the early years of life; it is also fair to assume that there is other tissue in the body, as for instance the lymph glands, which have the same function;

this function is mainly protective, however it may be explained. Removal of the tonsil, therefore, does not remove from the body any organ or tissue absolutely necessary to the system, but, if not diseased, its removal has left the individual with one defense less.

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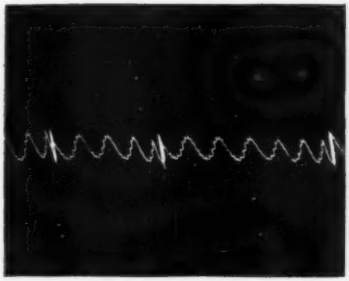
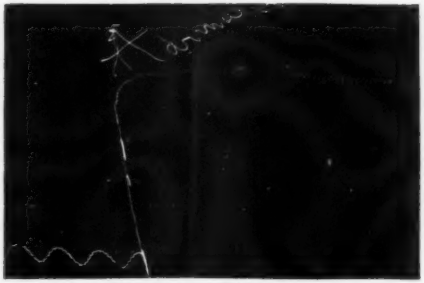


Tonsil Extracts.

Salt solution extract injected. Note the absence of change in curve.



Adrenal Extract.

Note great rise in curve caused by injection of adrenalin solution 1/10,000 in very small amount.

Copper Chlorid Extract.

Note lack of change in curve from normal after injection of tonsillar extract made by treatment of tissue with copper chlorid, and then removing the copper.

Extract Not Copper-free.

Note the fall in blood pressure caused by the presence of copper in the extract. When same solution is made free of copper the curve remains normal.

X.

SUBMUCOUS RESECTION OF THE NASAL SEPTUM IN THE SEMIRECUMBENT POSITION.

By William R. Butt, M. D.,

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To secure reasonably uniform success in all types of septal deflections by submucous resection, the following are essentials:

First—A knowledge of the anatomy of the septum—particularly that gained from studying it in relation to its embryologic development. (See a previous paper.)*

Second—An operating position and anesthesia which secure comfort to the patient and at the same time ease of manipulation to the operator; and in operating under cocain anesthesia, endeavoring to eliminate the personal equation of the patient as far as possible.

Third—Detailed methods of technic to overcome the mechanical difficulties.

The semirecumbent position, in my opinion, is ideal for nasal and oral operations done under local anesthetics. The position comprehends the patient lying on a table with the back raised at an angle of 45 degrees, and with the headpiece of the table made adjustable to a position comfortable alike to the patient and the operator. An electric bulb on an adjustable bracket or placed in a proper position on the wall, and an ordinary head mirror as a source of light, have proven most satisfactory, though a Freeman or Kirstein light have some advantages.

The toxic systemic effects of cocain and adrenalin with the patient in this position are incomparably less than those produced by these drugs on a patient in a sitting position. Since using this position, and the method of

*"Some Considerations in Reference to the Nasal Septum." The Laryngoscope, December, 1912.

anesthesia to be described, during the year past, the operation has not been interrupted by the patient in any case and there has been practically no complaint of pain or discomfort. The patient's head rests in a steady position against the table; he is at rest and comfortable and there is no necessity for hurry. The patient does not complain of stiff neck or grow weary, as he does from the strained positions made necessary in doing the operation in the sitting position. The headpiece of the table is raised to a position in which the operator, while standing, can work comfortably without stooping. If an assistant is employed, he can work at the opposite side of the table without interfering with the operator. The patient's head is kept rather to the side of the table from which the operator works. I now have an Allison table which permits this position for use in the office, and with the arrangements shown in the illustration the operation can be done without assistance. Two days' stay in the hospital, however, is much preferable when feasible.

The following method of local anesthesia has been highly satisfactory: First, both entire nasal fossæ are wiped over roughly once or twice with cotton wound applicators dipped in ten per cent cocain to produce general superficial insensibility throughout to further manipulation. In a few minutes, cotton tipped applicators dipped in full strength adrenalin are dipped, after the excessive moisture has been removed, in powdered cocain. This socalled "cocain-adrenalin mud" of Freer is massaged thoroughly into the entire septum on both sides. Two or three applicatorsful suffice. Then one per cent cocain in 1-10,000 adrenalin (which is made up roughly as desired by adding three drops of adrenalin to thirty drops of one per cent cocain solution) is injected hypodermically along the line where the incision is to be made and along the nasal floor. This last hypodermic injection secures anesthesia along the base of the ridge, which is usually a painful spot, and also in the skin margins, which are not easily reached by cocain which is rubbed in.

These three procedures usually secure entire anesthesia and blanching of the entire septum, so that no further applications are needed. However, after the first incision is made, if there is bleeding, full strength adrenalin solution is rubbed in the cut; but I believe that the success of the operation depends

considerably upon securing satisfactory anesthesia and ischemia at the outset before making the primary incision. This requires about fifteen minutes.

Since hearing of the death of a patient instantly following the hypodermic injection of ten minims of 1-1000 adrenalin into the nasal septum, reported by Dr. Freudenthal, I have never used adrenalin solution full strength hypodermically. I have myself observed severe symptoms of shock following the hypodermic administration of full strength adrenalin into the septum—despite the fact that this drug is recommended for combating shock.

The following details of technic are those which I have adopted from various sources: I make the incision described by Yankauer—usually on the side of the septum, which is convex anteriorly. An incision in the patient's left nostril has the advantage that the right hand is more easily used for the operating instrument, while a finger of the left hand can be inserted in the opposite nostril or used in holding the speculum, etc. If the deflection is very much to the right, however, the incision is made on that side, and in this case by bringing the left hand over the patient's face (while standing on the patient's right) the right hand is still free to do the actual dissecting, and a finger of the left hand may be inserted in the patient's left nostril.

The incision begins at the highest part of the septum in front of the deflection, and is carried down to the floor of the nose and partly across the nasal floor, practically into the skin of the vestibule. This incision also seems to me ideal. If each nasal fossa be represented as a right angled triangle, with the septum as the upright limb, then by this incision we lift the mucoperiosteal flap away from the vertical and horizontal limbs, allowing the loose flap to be pushed against the outer nasal wall (which represents the hypothenuse of the triangle). This gives a complete exposure of the septum and room for instruments without any stretching of the flap, as is necessary in a simple buttonhole incision. After the operation there is no retraction of this kind of a flap. The cut of the primary incision must be deep enough to go through the periosteum and perichondrium, but not necessarily at one stroke—the line of the incision may be gone over a second or third time, to make sure that it is down to the cartilage

and bone. A very helpful procedure in ascertaining whether the perichondrium has been cut through is to take the side of the point of the knife and scrape it along the line of incision until the cut edge of the perichondrium is seen. Then the cartilage is separated from the mucoperichondrium as far down as the vomer ridge, beyond which dull dissection cannot be carried, because the periosteum of the vomer here crosses from one side of the septum to the other. Freer's round knife and a sharp pointed spoon curette are useful in carrying the primary incision completely down to the bone along the lower part of the septum and into the nasal vestibule. Beginning now again in the primary incision near the nasal floor beneath the ridge, taking advantage of the fact that the periosteum is easily separated here, the thin dull Freer dissector is inserted under the periosteum here at the floor and worked up towards the top of the vomer ridge.

Now we have the entire flap lifted up from the convex side of the septum except at the single line along the vomer ridge, where it can be seen as a longitudinal band passing over the crest of the ridge. This is easily cut by a sharp dissector from below, often also assisted by scraping with a sharp spoon curette from above, with the cutting edge turned in toward the bone.

In getting through to the other side of the septum a little furrow is first scraped with the curette or point of the knife used sideways, a little deeper in the nose than the incision through the mucous membrane. Into this furrow the semi-convex end of a small (Potts') dissector is inserted with the convex side turned toward the opposite nostril, using a finger in the opposite nostril as a guide. With this technic of incising the cartilage a perforation is seldom made at this point. The cartilage is freed on the opposite side down to the vomer ridge, and the necessary cartilage is removed by swivel knife and flat forceps. The adherent point on this side along the crest of the ridge is freed by cutting with the sharp dissector from above and scraping with a sharp spoon curette turned in toward the bone, as described for the other side, and the periosteum is separated down to the nasal floor. Both flaps are now entirely free, and the cartilaginous septum removed. The bony ridge is cut along the floor with the Hurd forceps (which avoids rough fracturing) and the bone bitten out with

small forceps—the extreme anterior part being bitten out by holding the forceps perpendicular to the nasal floor; and sometimes by hooking the beaked end of the mastoid spoon curette around it and fracturing it forward.

The necessary portion of the perpendicular plate of the ethmoid is removed by fracturing it off with small strong double curette beaked forceps, an attempt being made to have the lines of fracture such that they will not be carried up into the cribriform plate. A bone cutting forceps is probably safer for removing the perpendicular plate, but those forceps on the market are too large and clumsy. That it is possible to open into the cribriform plate by the ordinary septum forceps used carelessly, I have demonstrated to myself on the cadaver. The suggestion has been made that opening the cribriform plate, though undesirable, is probably little more dangerous than exposing the dura in a mastoid operation, which is seldom followed by serious consequences.

I have used both cotton splints and gauze packing, and have had hematomata of the septum following both. These I believe were due to the fact that the packing was too tightly placed over the lower and anterior parts of the septum and over the mucous membrane incision, preventing escape of what blood might exude between the flaps after the operation. The ultimate results of these hematomata when opened and drained were practically as good in a couple of weeks as though none had occurred. However, in the first two cases of this kind encountered, these blood clots were not cleaned out, and when the patients passed from observation the hematomata had apparently organized and the nasal fossæ were more obstructed than before operated on.

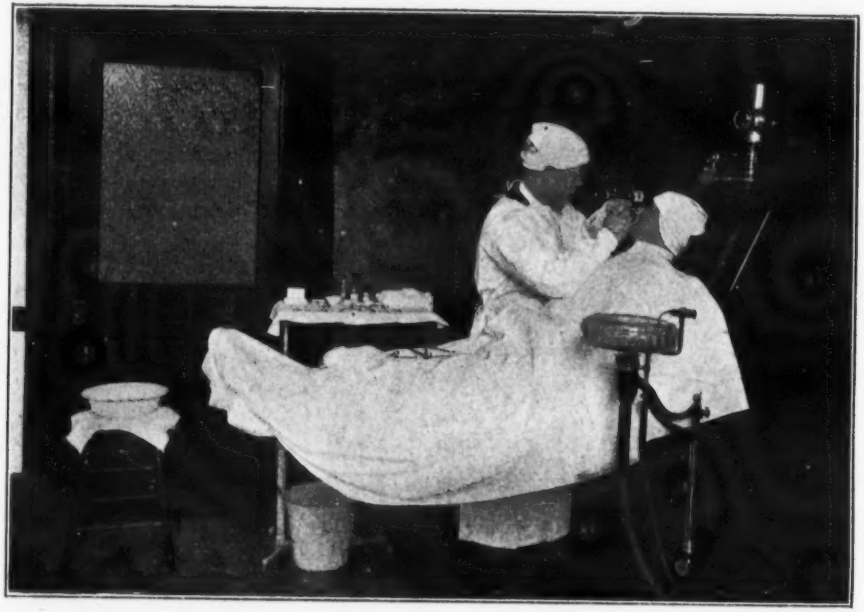
At present I use the cotton splints, packing both sides and placing them rather high, not bringing them farther anterior in the nose than the line of the primary incision in the mucous membrane. The packing is removed in from eighteen to twenty-four hours.

In two cases in young persons, where a general anesthetic was thought necessary, they were etherized lightly, then the septum was prepared as for local anesthesia, only sufficient ether being given to keep the patient asleep, depending upon the local anesthesia to abolish sensation. If it were safe to use adrenalin hypodermically in unlimited quantity, the

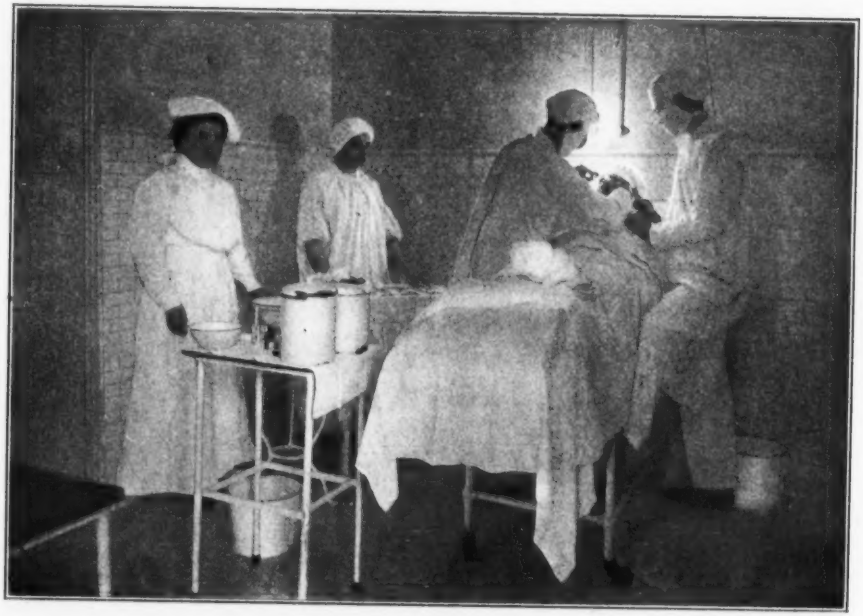
operation would be much easier under general anesthetics. In one of these two cases under ether the bleeding was well controlled and the operation easily done—in the other there was considerable bleeding, and the work had to be done largely by the sense of touch, which resulted in the loss of considerable mucous membrane on one side, but the septal deformity was satisfactorily corrected, and there was no perforation. In this case the pharyngeal tonsil was also removed before putting in the nasal packing. In another case, after the submucous resection was done under cocain anesthesia, ether anesthesia was used to remove the adenoid mass.

In operating under general anesthesia, the semirecumbent position has the disadvantage that mucus and blood, and also the tongue, tend to fall backward and obstruct the breathing. These tendencies can be overcome by inserting a postnasal tampon and keeping the tongue held forward with tongue forceps.

In conclusion, I express indebtedness to Dr. Wood, Dr. Babbitt and Dr. Butler for hospital opportunities for doing part of this work.



Office Arrangement for Septum Operation.



Hospital Arrangement for Septum Operation.

XI.

TREATMENT OF DEAFNESS BY REEDUCATING THE HEARING.

By Dr. Maurice,

Paris.

Three elements come into play in the mechanism of audition —the muscles, the nerves, the organs of transmission. If one of these elements is damaged, the hearing is affected.

The treatment is determined by these conditions. Until recently we have been very much occupied with the tympanic organs, the organs of transmission, the tympanum and ossicles, but too little with the labyrinth and the organ of Corti:

Let it be understood that I am not discussing absolute deafness, but the generality of cases, which includes all those persons attacked with hardness of hearing, more or lass accentuated, even quite advanced, but not complete deafness. So, we do not treat deafness, but impaired hearing. To reawaken a sense something must remain; we do not create, we invigorate.

I will not speak of the well known methods of retarding or lessening deafness, such as general treatment of the causal diathesis, nasal treatment, catheterization of the tube, bougieing, mobilization of the tympanum and ossicles, etc. I have been looking for a method of perfecting the results thus attained.

I have been preceded in this line by Urbantschitsch, Rousselot, Natier, Marage, Surguet, Villot and others, and a study of their methods has helped in establishing my conclusions.

The sound wave is the normal functional excitant of the ear. Its vibrations make the muscles of the middle ear tense, the hammer and stirrup to listen or to protect themselves. It keeps up the free articulatory play of the ossicles. It strikes

the organ of Corti and excites its functioning. The essence of the problem consists, then, in regulating, dosing, and exactly measuring the quantity and quality of sound wave necessary for this physiologic stimulus.

The method of Dr. Villot, which consists in exercises with an ear trumpet, is worthy of interest. It would be the method for a patient who has time to wait for improvement; it would not be a rapid, intensive reeducation.

To obtain the maximum of intensive reeducation, which I am discussing, we must have an apparatus for producing sound waves with certain desired characters. The tonality should vary at will and comprise the entire zone of the human voice.

The intensity of the sound should be modified according to the sensitiveness of the patient. One with dysacusia requires less intense sounds; another will stand the maximum.

Regularity of emission is quite important, since the intensity having been determined, we then have only to vary the tone.

Facility must be had for regulating the sounds reaching the ears, each independent of the other.

It is necessary that the vibrations produce a genuine sound massage of the organ, such as gives the ear a sense of titillation.

The principal note supercharged with numerous harmonics should give much resemblance to the human voice.

We could hardly ask more from an apparatus for reeducating the hearing, unless it is the telephonic transmission of the sounds obtained. In fact, the vibrating plate, placed close to the ear, imprisons between itself and the drum a cushion of air which transmits the entire vibration to that organ.

I am particularly pleased to state that I have succeeded in constructing an apparatus which fulfills the above conditions. I have christened it the "Kinesiphone," because of the vibratory massage effect combined with the emission of sounds.

In my apparatus the sounds are due to interruptions of the current in an electric resistance circuit on the phone wire, permitting regulation of the intensity. Tonality is obtained by an elastic vibrating plate, which augments the number of vibrations. To get a greater variety of sounds I have three plates, corresponding to low, medium, and high. The chords

are due to the phenomena of self-induction which take place in the circuit.

While we are on the subject of sense reeducation I want to say that I highly esteem Dr. Gernet's method, which consists in giving the muscles of the middle ear a progressive stimulation, a functional reeducation which is of the greatest utility for the end which we seek. I advise it systematically for all the patients that I treat, and especially for those who have retardation of auditory accommodation.

The best way of demonstrating the worth of a method is to show cases. From the large number which I have I will cite the principal ones. I will select them from all classes of deafness, for I affirm and will prove that the reeducation treatment applies to all forms; sclerosis of the tympanum (cases II and III), of the labyrinth (cases IV, V and VI), primary or rhinogenic sclerosis (case III), dry cicatricial otitis with atrophy of drum and ossicles (case I), etc.

The improvement obtained has been determined by measuring for the watch, whispered voice and spoken voice. The multitude of measurements eliminates error.

For the voice test I pronounce at the end of expiration words so chosen that their enunciation will not be forced or involuntarily amplified. The words which I use fall into two classes: low, isosonorous words and high isosonorous words. This classification comes from experienced phoneticians, such as Rousselot, Hermann, Zwaardemacker, Marage, and others.

The low note is generally less well heard by sclerosed thickened tympani, while the high note is less easily heard in labyrinth trouble with normal tympanum.

In the following reports I have taken the mean between the high and low, unless otherwise indicated.

Spoken voice is indicated by a straight line (————), whispered voice by a series of dashes (— — — — —), and the watch by dots (. . . . . .).

Case I.

Miss G., 18 years, neglected otorrhea had caused loss of tympanum and ossicles. I was able to dry the ears, but not to affect the hearing. Reeducative treatment alone has given a most interesting result.

WATCH.

	Right Ear.	Left Ear.
Before December 7, 1911	7 c. m.	3 c. m.
After six sittings, February 21, 1912	42 c. m.	8 c. m.

WHISPERED VOICE.

	Right Ear. Isosonal Words.		Left Ear. Isosonal Words.	
	Low.	High.	Low.	High.
Before	0.4 m.	0.8	0.12	0.35
After	7. m.	12.	5.	6.5

VOICE.

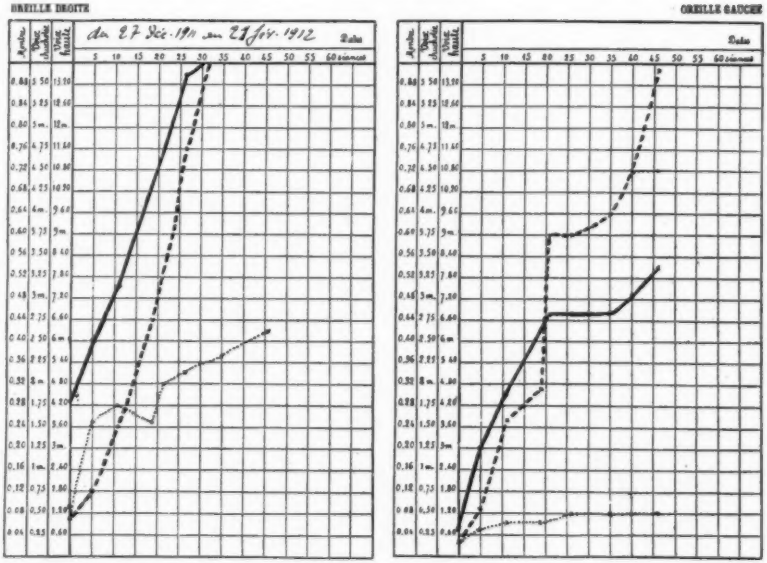
	Right Ear. Isosonal Words.		Left Ear. Isosonal Words.	
	Low.	High.	Low.	High.
Before	3.5 m.	5.	0.4	1.2
After	15. m.	15.+	6.5	9.

I defy any other method to produce such a result. The success can only be explained by mobilization of the cicatrices obstructing the labyrinthine windows, and by excitation of the organ of Corti which, however, was in good condition prior to treatment.

This young lady experienced transitory relief, before re-education, from destroying the cicatricial membrane obstructing the round window. This slight improvement disappeared in two or three days, when the cicatrix reformed.

When seen April 19th she heard very well, without any regression.

CASE I.


OREILLE DROITE
OREILLE GAUCHE
du 27 Déc. 1911 au 27 Fév. 1912
Dates
5 10 15 20 25 30 35 40 45 50 55 60 minutes

Case II.

Mr. D., aged 33, deaf since the age of 15, so that study was prevented. Diagnosis: Tympanic sclerosis, labyrinth unaffected. Previously treated by others and myself in the usual ways, especially catheterization, without result.

WATCH.

	Right Ear.	Left Ear.
December 21, 1911, before reeducation.....	1. c.	1. c.
January 30, 1912, after 69 treatments.....	19. c.	14. c.

WHISPERED VOICE.

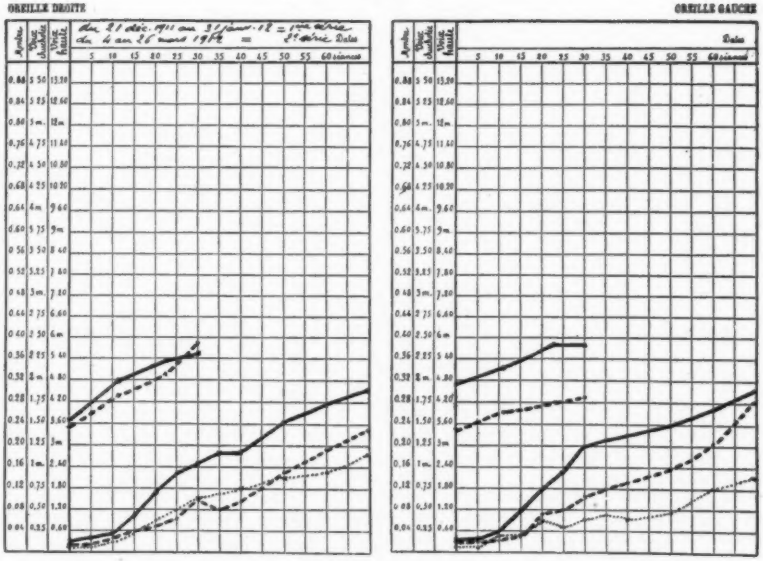
	Right Ear. Isosonal Words.		Left Ear. Isosonal Words.	
	Low.	High.	Low.	High.
Before	Ad aud.	0.18 m.	Ad aud.	0.18 m.
After	0.40 m.	2.50 m.	0.35 m.	3.20 m.

VOICE.

	Right Ear. Isosonal Words.		Left Ear. Isosonal Words.	
	Low.	High.	Low.	High.
Before	0.10 m.	0.60 m.	0.10 m.	0.60 m.
After	3. m.	6. m.	3. m.	6. m.

The result is particularly gratifying because of the duration of the trouble. The patient having demanded a quick cure, I give him thirty treatments from the 4th to the 26th of the month, which brought a little improvement, as may be seen on the chart (upper curve). As the patient lived in the province, I gave two treatments daily to gain time. The return of his hearing has enabled Mr. D. to change his business.

CASE II.


OREILLE DROITE
OREILLE GAUCHE
Voix basse
Voix chuchotée
Voix haute
5 10 15 20 25 30 35 40 45 50 55 60 secondes
0.88 5 50 13.20
0.84 5 25 12.60
0.80 5 m. 12 m
0.76 4 75 11.40
0.72 4 50 10.80
0.68 4 25 10.20
0.64 4 m. 9.60
0.60 3 75 9 m
0.56 3 50 8.40
0.52 3.25 7.80
0.48 3 m. 7.20
0.44 2 75 6.60
0.40 2 50 6 m
0.36 2 25 5.40
0.32 2 m. 4.80
0.28 1.75 4.20
0.24 1.50 3.60
0.20 1.25 3 m
0.16 1 m. 2.40
0.12 0.75 1.80
0.08 0.50 1.20
0.04 0.25 0.60

Case III.

My colleague, Dr. L., of Paris, aged 30, has tympanic sclerosis, probably rhinogenic, for the tube is atresic and he is subject to coryzas. Treated by me for six months he got no results, in spite of a thorough nasal treatment, insufflation of hot air, and vibratory massage. His condition grew worse from day to day until he could hear only with difficulty. I then tried reeducation.

WHISPERED VOICE.

	Right Ear. Isosonal Words.		Left Ear. Isosonal Words.	
Dec. 22, 1911—	Low.	High.	Low.	High.
Before reeducation	0.30 m.	0.80 m.	1. m.	7. m.
Feb. 7, 1912—				
After treatment	6. m.	7.50 m.	12. m.	15. +m.

VOICE.

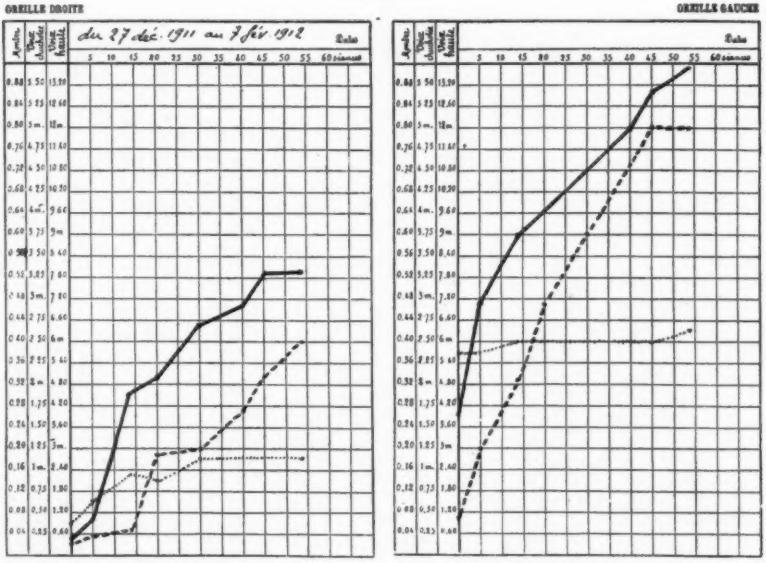
	Right Ear. Isosonal Words.		Left Ear. Isosonal Words.	
	Low.	High.	Low.	High.
Before	0.40 m.	1. m.	4. m.	8. m.
After	8. m.	13. m.	14. m.+	15. m.

Right hearing for the watch has increased in the same time from 6 cent. to 18, and the left from 28 to 42 only. Words in "ou" and "on," not perceived by the right ear, were heard at the end of treatment from 1.75 to 2. m. My colleague can really hear very well. I was particularly gratified with this result in a physician who could himself estimate the benefits of the method.

Seen March 22, 1912, the amelioration was maintained in spite of a cold which the patient then had. Audition for the watch had even increased on the left from 42 to 48 cm., although it had remained almost stationary during treatment.

The chart is traced for isosonal low words. Audition for whispered words is shown in the third column (that for voice), for whisper being heard almost as well as voice, the left tracing would have gone out after the eighteenth sitting if it had been inserted in its regular place (second column).

CASE III.



Case IV.

Mme. G., aged 44, sent to me by Dr. Hansen, of Paris. This lady could not hold conversation at a distance of 1.5 m. at the beginning of treatment, as Dr. Hansen and I confirmed. She had otitic adhesions with discharges from the labyrinth, and this trouble antedated three years. The drum was retracted, so I commenced by catheterizations, which were continued for a month or so. Her condition remaining the same, I attempted reeducation, after her hearing had been exactly measured.

WATCH.

	Right Ear.	Left Ear.
Dec. 22, 1911, before reeducation	2. c.	.35 c.
Jan. 18, 1912, after 24 treatments	22. c.	.40 c.

WHISPERED VOICE.

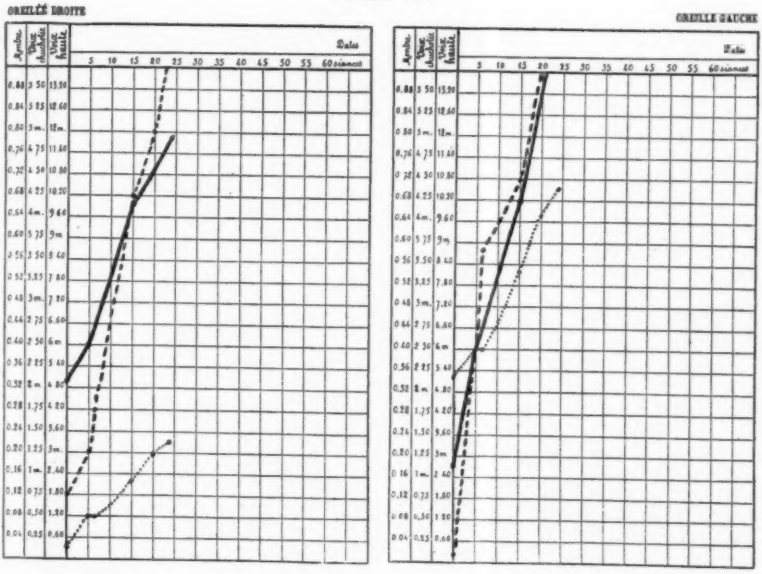
	Right Ear.		Left Ear.	
	Low Tones.	High Tones.	Low Tones.	High Tones.
Before	0. m.	35. m.	0.15 c.	0.01 c.
After	4. m.	50. m.	5. m.	12. m.

VOICE.

	Right Ear.		Left Ear.	
	Low Tones.	High Tones.	Low Tones.	High Tones.
Before	4. m.	6. m.	1.50 m.	4. m.
After	12. m.	12. m.	15. m.	15. m.

A rather stubborn eczema of the canal was very much improved by the kinesiphonic treatment, and that I attribute to the effect of the vibration. The eczema of the canal produced, by irritation from a branch of the pneumogastric, a morning cough which disappeared entirely after a few treatments. I desire to call attention to the hearing in the left ear of the low tones of the whispered voice, which has increased from 1 c. to 12 m. The patient when last seen had continued improving.

CASE IV.


OREILLE DROITE
OREILLE GAUCHE
Voix haute
Voix chuchotée
Voix haute
Voix chuchotée

Case V.

Mr. Ar., 52 years, found that his hearing, especially in the left ear, was gradually growing worse. I found, after examination, adhesive otitis, with involvement of the labyrinth; the drum was relatively large, and catheterizations had had no good effect. The involvement from the labyrinth discouraged me, but nevertheless I submit the results.

WATCH.

	Right Ear.	Left Ear.
December 22, 1911	10. c.	2. c.
January 10, 1912 (26 treatments)	35. c.	11. c.

WHISPERED VOICE.

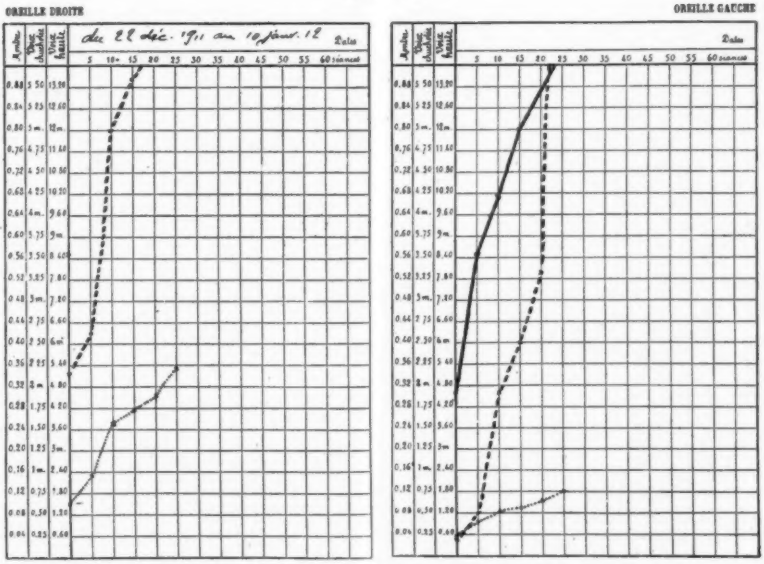
	Right Ear.		Left Ear.	
	Low Tones.	High Tones.	Low Tones.	High Tones.
Before	1. m. 80.	2. m. 50.	0. m. 30.	0. m. 10.
After	8. m.	13. m.	10. m.	8. m. 50.

VOICE.

Right Ear.	Left Ear.	
	Low Tones.	High Tones.
Impossible to measure because of its relatively good condition.	5 m.	2 m.
	Room too small to measure improvement. Call it 15 m.+	

It is the improvement in the whispered voice of which I shall make special mention. The patient's hearing was measured after the twenty-sixth treatment, as I stopped there, considering the result sufficient. When spoken to, Mr. A. made no distinction between the hearing of either ear, while before treatment he was obliged to place the speaker to his right ear.

CASE V.


OREILLE DROITE
OREILLE GAUCHE
Dates
5 10 15 20 25 30 35 40 45 50 55 60 secondes

Case VI.

Mr. U., fifty-five years old, was treated in 1908 by Dr. Archambaut for labyrinthic deafness which seemed to increase. He was hard of hearing from childhood; his father and two uncles were deaf. His deafness had increased very suddenly, as the result, he said, of a kiss on the ear. I thought this a special case, although the mercurial treatment had been of no avail. The catheterizations Dr. G— and I attempted did not help his hearing.

WHISPERED VOICE.

	Right Ear.		Left Ear.	
	Isosonal Words.		Isosonal Words.	
Jan. 3, 1912—	Low.	High.	Low.	High.
Before reeducation..	Not heard	Not heard	Not heard	Not heard
March 1, 1912—				
After 50 treatments..	0 m. 30	0 m. 25	0 m. 25	0 m. 25

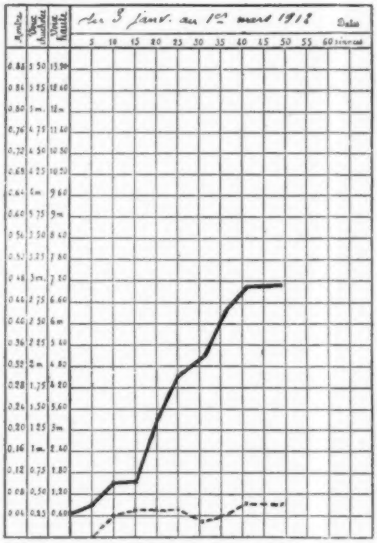
VOICE.

	Right Ear.		Left Ear.	
	Isosonal Words.		Isosonal Words.	
	Low.	High.	Low.	High.
Before	0 m. 60	0 m. 80	0 m. 80	0 m. 80
After	7 m.	7 m.	6 m. 80	6 m.

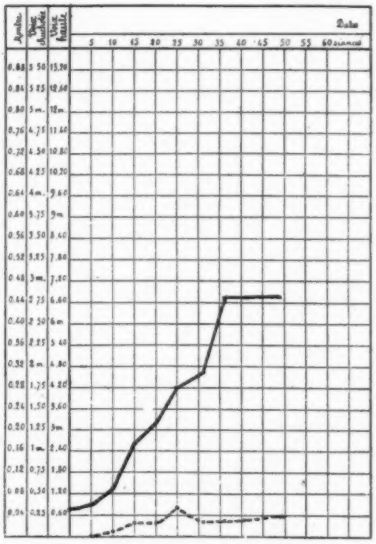
The watch was not heard. The patient at first stood the acoustic exercises with difficulty; soon he very rapidly accustomed himself to the maximum.

CASE VI.

ORELLE DROITE



ORELLE GAUCHE



The conclusion to be drawn from the observations above is that no other method is capable of producing these results, since all the patients noted had tried the standard treatments in use.

I am able to state that all kinds of deafness can be ameliorated, as well as one can claim that tuberculosis in the first stages is curable. There are no rules without exceptions, and in kinesiphonic reeducation, the exceptions are rare—one in thirty, from my actual observations.

In conclusion, I wish to attempt to explain the process of improvement and then of the cure.

Light massage produces, by mobilization of a physiologic sort, thickened tympanum and ankylosis of the ossicles. Pneumatic massage accomplishes very little, because these organs are intended to vibrate at a high degree of speed and not at the reduced speed of the masseur of Delstanche or the electromotor of Breitung.

Gentle vibration produces on the membranes of the drums of Narey molecular modifications which help them and which permit them by usage to make better inscriptions. These modifications are found in the case of a violin in which the sound is most harmonious after much training.

The tympanum should act in the same manner, after several treatments by reeducation.

The treatment succeeds in producing a vasodilatation visible to the otoscope, a vasodilatation whose fine result must be most useful to an organ almost destroyed. The return of the ceruminous secretion, the amelioration of certain dry eczemas of the canal, amply prove it and allow us to extend to the middle and inner ears what is ascertained in the external ear.

Powerful sounds given in the right way stimulate the indifferent hearing of a deaf person. Most deaf people are careless of hearing because they do not use their ears. Ultrarapid recoveries can hardly be explained except in this way. As soon as one does not hear the human voice well, he does not hear any longer because it tires him to listen; he isolates himself, as far as his hearing is concerned, until he relapses into a deafness more and more complete.

The stimulation of many fibers and cells of the organ of Corti by sounds befitting their number and their function can only reawaken an organ on the road to atrophy, to sclerosis, to functional paralysis.

Moreover, we can imagine that violent vibration favors the contact of cylindrical terminations and cells of the organ of Corti, as it produces between the carbon granules in a microphonic apparatus.

All these reasons can explain my results.

The improvement has been maintained and has the tendency to continue, for the patient, by himself, pursued his physiologic training. In the meanwhile he listens to noises and continues the reeducation.

If the cause of deafness persists in making the affection evolve, a loss of hearing is not manifested when the cause is not reached by reeducation; in that case it is good to attempt at the end of a while, a new one. In advising the patient to perform the exercises which Dr. Villot values, by means of an acoustic horn which is not measured, in most cases a non-receding amelioration is obtained.

The cases which I cite most strongly and which are quite controllable show unquestionably the foundation of the method and the hopes there are to expect of it. I am entirely of the opinion of my confreres who wish to judge de visu the truth of what I have put forth.

SOCIETY PROCEEDINGS.

NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLOGY.

Meeting of November 8, 1912.

Case Showing Graphite Stains in the Membrana Tympani.

DR. A. P. VOISLAWSKY: In February, 1911, J. C., four years of age, was brought to my office suffering from an acute discharge of the left ear, due to a traumatic injury of the membrana tympani, self-inflicted with a toy gun having a lead pencil as its projectile. The little patient placed the gun in the left auditory canal and pulled the trigger. The projectile entered the membrana tympani in the anterior inferior angle, and for six days there was an acute discharge which promptly yielded to treatment. One year and a half later, on examination, the hearing was normal, but at the point of entrance was a well marked graphite stain, showing that at the time of entrance some particles of the lead imbedded themselves in the membrana tympani.

A Case of Tumor in the Cerebellar Pontine Angle, Presenting Early Symptoms of Deafness and Vertigo. Preliminary Report of Autopsy Findings.

BY JOHN ALEXANDER ROBINSON, M. D.

DISCUSSION.

DR. DIXON said that it was difficult to state positively whether the case was an echinococcus cyst or not. Examination of the brain perhaps would have enabled one to be more positive. It certainly was a cyst, but what form it was not possible to state exactly.

DR. DENCH had seen the case with Dr. Robinson, and wished to emphasize the importance of a thorough otologic examination in all cases in which some lesion below the tentorium is suspected. He continually sees a number of cases in his service in the Neurological Institute, and finds the work of the otologist in making a careful examination to be of distinct value in localizing the exact position of the lesion. He would impress on all otologists the importance of a careful examination when tumors are suspected.

DR. MCKERNON had been interested in observing how long these patients would live after simple decompression operations. He had seen two cases with Dr. Peterson which differed from Dr. Robinson's cases in having less hearing on affected sides, and tinnitus was more pronounced. One was operated upon by Dr. Cushing, a decompression operation being performed. The patient lived for eight months and four days after operation, suffering with a large brain hernia. The second died four and a half months after Dr. Cushing's operation, with a tremendous brain hernia present. These cases simply show how long on an everage such patients live after decompression operation.

DR. WILSON asked for how long after the decompression operation did the optic neuritis remain cleared up.

DR. MCKERNON replied that the neuritis improved for about six weeks, then increased with greater severity.

Some Systemic Factors in the Etiology of Progressive Deafness.

DR. J. A. STUCKY, in introducing this subject, deplored the fact that these conditions still stand impregnable to the attacks of the otologist, in that so little can be done for their relief and that so little is known of their inherent etiology, but extended the hope that in working in close relation with the internist and the neurologist, more can be known of the causation and more done to inhibit their progress, even though no cure is found.

Two classes of cases were cited, those of a nonexudative otitis media and those of otosclerosis, both resulting in progressive deafness. A definite pathology exists from each condition.

In the nonexudative otitis media there is little change in the

membrana tympani, slight retraction, atrophy, opacity, or small calcareous deposits, the eustachian tube showing no change to account for the condition, and because of the progress of the deafness, it must be concluded that ankylosis is present in the ossicular joints and fenestral structures to a greater or less degree.

There may or may not be an associated nasopharyngeal condition. The etiology of these conditions still remains the shuttlecock of otology, but all agree that the cause is nonspecific, but due to some cause foreign to the ear itself. The drum membrane shows no deviation from normal, unless it be complicated by a nonexudative otitis.

In otosclerosis the disease begins in the bony capsule of the labyrinth, especially near the oval window, rarely at the fenestra rotunda. In the regions so affected the osseous tissues become vascular, giant cells and osteoblasts are present, forming overgrowth of spongy tissue, whereby the foot of the stapes and annular ligament are involved, with a resulting ankylosis. The function of the stapes is thereby interfered with or abolished.

Some victims of tuberculosis, syphilis, nephritis, rheumatism, organic heart and other circulatory conditions are affected secondarily with one or the other types of progressive deafness, due to circulatory changes in the ear, or to a hyperemia because of high arterial tension.

Most especially does the author emphasize in all cases treated by him, the close relation of disturbed metabolism and toxin absorption produced by improper food and drink, these toxins entering the blood and so causing disturbance in the ear, the point of least resistance in those so affected.

As food, drugs, toxin, and alcohol changed by the same elements that each victim so afflicted asserts his or her idiosyncrasy to the individual poison to which this susceptibility exists, so in brief the assumption that the changes are due, to a large extent, to autointoxication and other toxic agents, predicts that future findings will support this contention. Heredity, only in that like conditions, like food, apply to the child as they do to the parent.

Nonexudative otitis media and otosclerosis are related only symptomatically, those of the former often merging into those of the latter.

In the presence of slight tinnitus and vertigo, with a history of varying degrees of deafness aggravated by any systemic or psychic disturbance which unbalances in any way the circulatory apparatus of the labyrinth, accompanied by the slight pathologic changes in the membrana tympani referred to above, one must consider the probability of the nonexudative otitis media, provided they do not respond to inflation and local treatment and are not caused by some nasopharyngeal or eustachian tube derangement. By Weber's test the bone conduction is increased, except in old persons, when it will be found decreased in intensity. Most especially should one look for some systemic disturbance.

Subjectively the symptoms of otosclerosis are those above, but intense vertigo occurs less frequently and seldom in aggravated form. Bezold's symptom complex is found, increased bone conduction and negative Rinné with elevation of tone limit. The membrana tympani is practically unchanged, unless complicated by nonexudative otitis media.

The following case reports are incorporated:

Nonexudative otitis media, eleven cases, nine males, two females, varying from 23 to 68 years of age, with hearing 25 to 50 per cent below normal, following a gradual diminution for one to ten years.

These cases were improved 10 to 30 per cent by strict regime of diet and no alcohol, together with alterative remedies. Occasional exacerbation occurs when overindulgence in eating or drinking occurs.

In seven cases of otosclerosis, three males and four females, five of which cases were seen by several prominent otologists and a very unfavorable prognosis given, especially those in whom an hereditary element existed, the ages ranged from 22 to 47 years, with 10 to 35 per cent loss of hearing power. By the same treatment three regained 15 to 20 per cent, and the other four, who were worse and at more advanced age, regained 2 to 5 per cent. The diagnosis in both of these series of cases was made after careful and frequent observations.

Just what systemic derangements cause progressive deafness, the author cannot say, but places rheumatism and arteriosclerosis, from whatever cause, as most important.

All cases were much aggravated by any disturbance of metabolism, no matter how brought about.

The treatment of progressive deafness advocated by the author is, briefly: assist elimination, get rid of any toxic substance by stopping intestinal absorption of toxin, as nearly as possible preserve a normal tissue metabolism.

To be most emphasized is the fact that the two types of progressive deafness can be differentiated, though closely related. Whichever are intensely aggravated by any disturbance of metabolism and which can be improved, stayed in their progress and often in a marked degree relieved by careful and persistent treatment by use of a careful dietary and alterative treatment.

DISCUSSION.

Dr. McKernon said that he did not feel qualified to discuss Dr. Stucky's paper. He thought, however, that the benefit which Dr. Stucky's patients derived was twofold—first, from an increased standard of health; and second, he did carry out a certain amount of local treatment, which was the secret of his success. In any aural disease the promotion of the general standard of health influences the aural disease favorably.

Dr. Stucky had spoken of gastrointestinal types of this condition. All have seen this in otitis media cases which have vertigo. Here, treatment directed to the gastrointestinal tract produces good results, while local treatment is not of much value. In other cases of otitis media, however, no such happy results are obtained. The cases suffering from vertigo are not improved if the symptoms are due to disturbance of circulation. Here, again, the treatment must be directed to the cause.

In other otosclerotic types the history shows no trouble with the nutrition or circulation, no gastrointestinal disturbance. The patients deny these, and yet they have otosclerosis. While Dr. Stucky had not spoken of these cases, the otologists owe them attention. There is a general tendency that local treatment is not of much avail. Dr. McKernon said, however, that he believed in these cases of slowly developing otosclerosis, local treatment applied to the tube, ear, nose, or nasopharynx can accomplish a great deal, and perhaps arrest the disease for some time. Especially was this true when general systemic treatment was used in conjunction with the local. A large series of cases coming under his observation

during the past few years has tended to confirm him in this opinion. It is difficult to say what is the etiologic factor in otosclerosis. The thanks of the section were certainly due to Dr. Stucky for bringing this subject before the members.

DR. DENCH thought Dr. Stucky's paper very opportune, as the members of the section had been accustomed to hearing so large a proportion of surgical papers. One point especially worthy of notice was that these patients usually come under close observation only after the second or good ear has become more or less seriously involved. We are naturally endowed with so much more hearing than is ordinarily required, that small degrees of impairment indicating the inception of disease go unrecognized. The patient may not even know that there is profound impairment of hearing in one ear, and it is only when the other or good ear becomes much affected that he applies for relief. It would seem to be most desirable that the family physician should test the hearing of his patients at least once a year, to detect the beginning of any aural disease. If such patients came under observation early, diet and other general measures would do more good.

Dr. Dench said that he believed vertigo was a common symptom of otosclerosis. At the present time it seems difficult to know just which cases do have otosclerosis—whether impairment of hearing with normal middle ear and labyrinthine changes constitute it, or whether labyrinthine changes alone are present. If these middle ear conditions do exist, they must, for the most part, involve the oval and round windows.

Dr. Neumann, of Vienna, during a number of years, had found at autopsy only fourteen cases of otosclerosis. This would emphasize the fact that the disease is not so common as is generally supposed.

He wished to confirm and emphasize Dr. McKernon's remarks on the value of local treatment. He has seen improvement occur from local treatment in a large number of cases, as shown by subsequent tests for hearing, including that with tuning forks—the lower tone limit having become lower. This surely shows the value of local treatment. He believes in proper systemic treatment when indicated, but the value of local treatment should not be overlooked. He has made the diagnosis by tuning fork and other tests, and yet treat-

ment has resulted in great improvement. These patients should be treated until it is demonstrated that treatment is of no avail.

Regarding the importance of systemic conditions, the ear is the organ of least resistance in these cases, and therefore becomes involved, and in such cases local treatment combined with proper general treatment should do good. Strenuous living seems to have a great deal to do with the causation of progressive impairment of hearing.

Dr. T. P. Berens thanked Dr. Stucky for calling attention in so able a manner to this much neglected phase of otosclerosis. Many cases of otosclerosis, accepting Dr. Stucky's definition, had been benefited by correction of faulty metabolism. During the past ten years he (Dr. Berens) had referred many cases to internists with marked benefit; particularly in preventing the only too frequent acute exacerbations of the disease. He believes that otosclerosis, as defined by Dr. Stucky, is in a great many ways similar to arteriosclerosis, and that its treatment should be conducted along the same lines as the latter.

Dr. Duel said that one must distinguish between cases of otosclerosis and cases of catarrhal otitis media. In his own experience he had come to regard as cases of otosclerosis only those with definite history of one or more ancestors who, like them, in early adult life (twenty to thirty years of age) had begun to develop progresive deafness. In his experience, local treatment in such cases had usually proved ineffectual. But in the socalled catarrhal type, in which the impairment had been due to some lesion in the conducting mechanism, improvement had usually resulted from vigorous local treatment. We have not yet definitely agreed as to all the causes of otosclerosis, but results forced nearly all to admit that treatment was incidental. He had observed some cases continuously for seventeen years, which had received a great deal of local treatment. He did not think that the local treatment had to any degree influenced the change in hearing which has taken place, though the moral support of such treatment might have been of value. It was difficult to say with any certainty whether or not such cases are influenced for the better by any local treatment.

Dr. Stucky's results with little or no local treatment seemed

to be as good as any one could show following vigorous treatment. Dr. Duel then cited the case of a patient whom he had seen two days previously, after a long interval. She had first come under his observation fourteen years before. He had "strenuously" treated her for several years; then two other otologists had treated her for several years; then she had been under the care of a Christian Scientist for several years more—and today her hearing was practically the same as it had been fourteen years ago.

The change in the labyrinthine capsule seemed to go on, despite all efforts to check it; in some cases, rapidly; in some, very slowly; in others, imperceptibly. If we can learn enough to influence otosclerosis by change in mode of life, etc., or find some internal treatment to check its advance, something may be done in generations to eliminate the disease. Perhaps something could be done by eugenics. He firmly believed that it was only through control of marriage of otosclerotics that the disease could ever be blotted out.

Dr. Hays said that one point of importance had not been brought out in the discussion. These patients often apply for relief when they are very deaf and are beyond the treatment of the cause of the condition. Certain connective tissue changes have taken place within the middle ear, similar to those which take place in cirrhosis of the liver and locomotor ataxia. It seemed evident that although much can be accomplished by general treatment, still a great deal of local treatment must be employed in order to overcome some of the connective tissue changes.

Dr. Sheppard wondered what the various gentlemen meant when they spoke of cases of "real otosclerosis." Our German colleagues have shown that Bezold's triad is not necessary for a diagnosis of otosclerosis. A review of the literature preparatory to writing a paper on the subject last spring, made it clear to him that he did not know what it was, and induced him to believe that no one else did. We clearly need more autopsy work. Is it due to disease of the middle or internal ear? Does it consist of Bezold's triad, with a normal drum and middle ear? One German writer on the subject found that a large percentage of his cases did not show Bezold's triad, but rather auditory nerve deafness. He agreed with Dr. Stucky as to the lack of value of local treatment.

If the middle ear, tube, nose, and nasopharynx are normal, why treat them? He has had no considerable number of cases in which relief was brought about by changes in metabolism, as cases with bad metabolism have abnormal mucous membranes, and are therefore not cases of otosclerosis. We should not discourage these patients, but should do our best by building them up to perhaps hold some of them stationary. As to eugenics, he had had two propositions put up to him. First, the family physician had referred to him a young woman who had otosclerosis and was sterile after several years of married life. The doctor was being urged to do some operation to remove her sterility, and the question arose whether it would be advisable, in view of the fact of her having otosclerosis. He advised letting her alone. The second instance was that of a woman who had otosclerosis (her mother also having had it) and who wished to get married. The question arose whether her hearing would get worse, and whether it might be transmitted to possible offspring. He had felt compelled to admit that both of these things might happen.

Dr. Stucky, in closing the discussion, expressed his appreciation of the free and candid discussion of his paper.

Replying to Dr. McKernon, he said that the vertigo spoken of in these cases was undoubtedly due to some circulatory disturbance, and that this disturbance was due to faulty metabolism. This was a point which he wished to emphasize, and which must be corrected. He had frequently seen cases in which the urine tests and microscopic examinations showed nothing wrong and the blood pressure was normal. One case had been examined by the internist and himself carefully at different times, and at no time could they discover any possible cause of the progressive deafness; a few months later the disturbing factor was discovered by a neurologist to be an emotional perversion. After several months of treatment by the neurologist this was relieved, and the patient slowly showed evidence of improvement, in two years the hearing being improved as much as five per cent.

There are periods in all these cases when there is a sudden decrease in the loss of hearing power, and these periods can be likened to the explosive attacks of gout. In many gouty cases there is a normal condition of the urine with slight variance in the blood pressure, and the acute attack of the gout

is accompanied by sneezing and all the evidences of acute rhinitis. Both the rhinitis and the gouty pains are speedily relieved as soon as the secretions are hyperalkalized and the alimentary canal is emptied and cleaned.

Dr. Stucky said that he had not intended to convey the impression that no local treatment was ever indicated in these cases, but wished to emphasize the point that he gives no local treatment in the way of inflations, vibration, or electricity unless there are positive indications for the same.

He quite agreed with Dr. Dench, in saying that there may be vertigo in the nonexudative catarrhal otitis media as well as in otosclerosis, and expressed his regret that Dr. Dench had not explained how he accounted for its existence in such conditions. If we exclude all possible localized causes of the vertigo and tinnitus, there being no evidence of middle ear, eustachian tube, or nasopharyngeal diseases, then the cause must be systemic. At any rate, in his own hands the greatest relief of these two conditions had resulted from systemic treatment. Probably no otologist would question the fact that there was danger of overtreatment by inflation and mechanical manipulation.

He quite agreed with what Dr. Duel had said, and had only referred to pregnancy, heredity, and eugenics in the paper as possible causes of otosclerosis. Personally, he had not much faith in the hereditary factor. One might inherit a tendency to this disease, but not the disease itself; and in some cases of this evidence of faulty metabolism in the child born of a parent who had otosclerosis, he believed the cause of the manifestation to be because until adolescence the child lives very much the life lived by the parent, eating the same kind of food, and with environments tending to cause faulty metabolism. He said. further, that if we begin early enough with a child who has an hereditary tendency to this disease—as he had been able to do with four cases within the last twenty years—that he believed they could be prevented from developing the disease. Pregnancy and parturition for the time being lower the vitality, and for the reason that "a chain is no stronger than its weakest link," the ear, if previously weakened, begins to manifest evidence of the disease. In the series of cases presented in the paper, three of them had otosclerosis and had borne children, one having had six chil-

dren, another four, and the third two. Pregnancy and parturition did not increase the deafness; on the contrary, there had been a slight improvement in hearing.

He expressed his indebtedness to Dr. Duel for the very appropriate phrase, "strenuous treatment," which in his own hands had done more harm than good. The improvement noticed from change of climate, rest and outdoor life was the result of arrest of imperfect metabolism, caused by living a more normal life.

Dr. Kerrison had summarized the discussion so thoroughly that it was needless to add anything to his excellent comments. Doubtless all present agreed with Dr. Sheppard when he said that "We all know so little about the disease that we are in danger of becoming nihilistic." Rather than do that, however, we should not only hold out hope to our patients, but be ourselves filled with the greater hope and determination to ascertain the cause of the disease and devise a mode of life and plan of treatment which would remove it. When we show the same interest and determination in dealing with cases of nonexudative otitis media and otosclerosis that we do with syphilitic and tubercular patients, and are willing to give them the same time, care and perseverance, we will begin to get results. Certainly nothing can be promised in these cases in less than a year's treatment. Each case is a law unto itself. The mode of living of the patient must be regulated as well as the diet. Reconstructive alteratives must be persisted in for a long time. Internal secretions must be regulated by such remedies as will accomplish these. In a word, our efforts must be directed to changing the faulty metabolism which results in these diseases into a normal metabolism which will relieve them. That progress has been made along this line and that a greater progress will be made in the future, there was little doubt. At any rate, in the treatment followed in the cases reported, and in others still under observation, in the long self-denial and rigid observance of certain rules and regulations, the patient has everything to gain and nothing to lose; and of those who are impressed with the deprivation and sacrifice which the future brings with defective hearing, a few are willing to abide by the rigid discipline which in the majority of instances gives such relief and encouragement that they are unwilling to return to the strenuous abnormal method of living.

NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLOGY.

Meeting of December 13, 1912.

Paper: Vertigo: A Clinical and Therapeutic Study.*

By Mr. Richard Lake, F. R. C. S.,

London.

Dr. Kerrison said that Mr. Lake's name was so closely associated with his pioneer surgical work for the relief of vertigo, that many of his auditors had probably expected that his paper would deal very largely with a discussion of surgical cases. Instead, he had contented himself with a very guarded and conservative statement as to the indications for surgical intervention in these cases, and had occupied his time very largely with a broad and philosophic discussion of the various causes of vertigo.

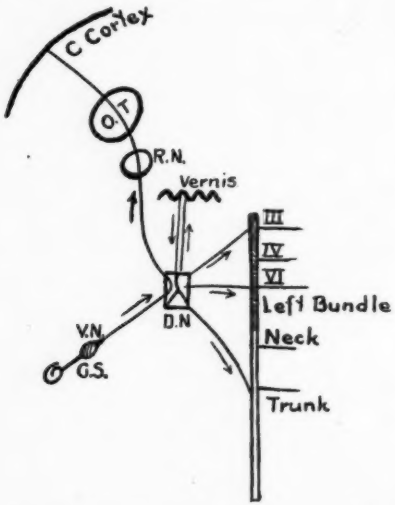
DISCUSSION.

Dr. Charles L. Dana said that he felt something like an intruder in the discussion, especially after listening to Mr. Lake's admirably presented paper, which contained so many points worthy of discussion and remark, and which he personally felt unable to criticise. He expressed his appreciation of the invitation to join in discussion of the subject, and was the more inclined to accept it because of his own rather definite views in regard to vertigo. He was interested to know whether these views were sound and would stand criticism.

It has been his belief that all vertigo is a vestibular affair. Vertigo is not always aural, but it always has to do with a disturbance in the function of the vestibular mechanism. Vertigo is a kind of neuralgia or paresthesia of the eighth nerve. The vestibular part of this nerve with its central tracts and connections make a very complex mechanism. The vestibular fibers originate in the ganglion of Scarpa and sup-

*See page 970, December, 1912, number of Annals.

ply the semicircular canals; then the nerve goes to the three vestibular nuclei in the medulla, which form its most important central station and a primary vestibular center; from there these nuclei send out axites in several directions. Some pass through the longitudinal bundle to the nuclei of the oculomotor nerves. Some few fibers pass up to the vermis of the cerebellum, but the connection here is very slight. The cerebellum is not the organ of the space sense, as was once



The vestibular nerve and its connections. The mechanism of equilibrium tonus is the V. N., vestibular nerve. D. N.—Deiter's and other primary nuclei. Nucleus in vermis. L. B.—Longitudinal bundle and its connections with oculomotor nerves and anterior horns. The sensations of vertigo are sent up through the red nucleus and optic thalamus to the cortex.

thought. The vestibular nuclei send afferent fibers to the red nucleus, and thence on to the optic thalamus, thence to the cortex of the brain. We do not know that the anatomic and physiologic relations cease there, for there are tracts from the cortex passing down to the pons and cerebellum, forming thus a circuit; so that in order to keep ourselves straight in the world, we have to play upon a very important and elaborate mechanism. There is not a moment in our lives when we are

not doing something to keep ourselves properly balanced in space.

The vestibular tracts are all afferent; they form a sensory nerve which carries impulses into the brain. When this is irritated, it causes a disturbance of sensation which is analogous to a pain or dysethesia, and which we call vertigo. This sensation is to the vestibular nerve and its central tracts and stations what pain is to the trigeminal nerve, and that is the basis on which we can explain the phenomena of vertigo. Just as the trigeminal nerve can be stimulated and pain be aroused by irritation of the peripheral part or of the roots, of the Gasserian ganglion, or of the central tracts, so the vestibular nerve can be irritated in the ear and labyrinth, in the vestibular root, the three vestibular ganglia and in its more central parts, thus arousing sensations of vertigo.

Dr. Dana said that while there is a large number of cases of aural vertigo, there is a much larger number of cases of vestibular vertigo, not at all aural. In neurologic and general medical practice we see ten cases of severe vertigo in which the ear is not the slightest degree involved, to one case in which the trouble is distinctly aural—at least that was his experience.

Most of the nonaural cases of vertigo can be explained on the theory of disturbance of the more central part of the vestibular mechanism. There is a disease known as migraine, which is a periodical sensory neurosis due to discharges from the cerebral cortex.

Sometimes instead of having migraine, a person will have an attack of vertigo. The speaker said that he had had a number of patients with attacks of this kind of migraine—vertiginous attacks—with Meniere's syndrome taking the place of the headache; other cases had vertiginous attacks lasting several hours, followed by severe pain and the sickness of migraine; in other words, the vertigo was part of the aura of migraine, just as zigzag lights are part of the aura. That would suggest that some forms of vertigo are cortical and due to cortical discharges. There is also epileptic vertigo which is known to be cortical.

There are some organic nervous diseases in which vertigo is one of the common characteristic symptoms; one of these is multiple sclerosis. In this disease the pons and medulla are very often affected, perhaps more so than any other part

of the central nervous system; and here the central paths of the vestibular nerve are affected. This theory of vertigo has been offered in cases that came to his clinic, and he has much simplified the explanation of the phenomena.

In regard to some of the forms of vertigo which are not aural, but which are very striking in their characteristics and have very distinct clinical syndromes, one form appears to be like a discharge from the cerebellar cortex. The cerebellum is not a center of the vestibular nerve, but the cerebellar nuclei do send fibers down to the vestibular nuclei, and the cerebellum is thus in close connection with the nuclei of the vestibular nerve; therefore it seems a reasonable theory that in these attacks where there is cerebellar disease and vertiginous attacks, the latter are due to the action of the cerebellar discharge upon the vestibular nuclei.

There are some interesting clinical facts regarding vertigo which are of special value to the neurologist. There is a condition which is sometimes aural, more often not, in which the patient does not have vertigo, but has the sensation that he is going to have it; a sense of insecure equilibrium, a sense of uncertain balance so great that the patient calls a cab and goes home for fear of falling in the street. Analysis shows these cases to be psychical in character; so that we have a vertigo of a type which may be called psychic, or, if you wish, hysterical.

Another form of vertigo which may have something to do with aural conditions is chronic or permanent vertigo. A patient of the age of twenty or thirty becomes dizzy and continues so for years during waking hours; this is not an objective vertigo, but a subjective one. The patient may go about attending to his duties, or may even play lawn tennis, but has always a certain degree of dizziness. That is known as permanent vertigo. This form sometimes runs through families. It has been described by Oppenheim.

The doctor said that he had listened to Mr. Lake's paper with great interest, and would like to discuss it at length. One point had much impressed him. Recently he had been told of a case of aural vertigo of a severe type in which it appeared there had been a hemorrhage into the labyrinth. The patient got well. There was no history, so far as was known, of syphilis, but two and a half years later the man had a general paresis. In his own experience, quite a large

percentage of cases of aural vertigo had been of specific origin. Mr. Lake had included paresis in his list, but had put very slight emphasis upon it, whereas it would seem to be a very important cause.

Dr. McKernon said that he had nothing to add to the paper, and only wished to express his hearty approval of it. The classification which Mr. Lake had made was admirable, for several reasons. In the first place, it was simple, and in the second, it could be easily applied; the cases could easily be grouped and followed.

Then he had been forcibly struck by the very small number of operations which Mr. Lake had found necessary to perform in the number of cases of distinct labyrinthine disease which he saw. This was very significant. Since taking up this work on the labyrinth and studying it more closely, he has come to the belief that at the present time—as well as in the past—there is too much indiscriminate exploration of the labyrinth; the labyrinth is opened too frequently, without definite and positive indications.

One very interesting point which Mr. Lake had brought out was the necessity for taking the blood pressure. He had also made another important point, i. e., that vertigo is a symptom and not a disease, and that its relation to other systemic conditions must be taken into consideration. He had also spoken of the advisability of not operating in cases where the hearing was good. This was in line with the views held in this country at the present day.

It was very interesting to notice the association of the low blood pressure with the hypersensitive vestibular apparatus. That emphasizes to a much greater extent than we have probably taken into consideration, the advisability of taking the blood pressure in all cases of labyrinthine disease when they first apply for examination.

Dr. McKernon said that among the several causes which Mr. Lake had mentioned, he had not heard him refer to exostosis of the external auditory canal. He himself knew of several cases in which these had been present and had caused vertigo. He also mentioned several cases coming under his observation of men addicted to the use of alcohol to a great extent. He had seen several such cases accompanied with labyrinthine hemorrhage and followed by complete and permanent loss of hearing.

Mr. Lake had also spoken of the lack of danger in doing the operation upon the labyrinth. He was certainly to be congratulated upon the results reported, and in his hands the operation may be devoid of danger, but in operating upon the labyrinth with either suppurative or nonsuppurative conditions, it has to be done with a view of danger in the future from complications arising.

Mr. Lake had also spoken of pilocarpin being given for two weeks. In this country it has been the habit to give it over a prolonged period of time, usually six weeks. He had also spoken of the first symptoms of arteriosclerosis in certain cases being vertigo. This was a very important point, and should lead to a more thorough investigation of these cases from a systemic standpoint than we have been giving them.

In speaking of the drugs used for this disease, Mr. Lake had not mentioned one which in Dr. McKernon's experience with cases of vertigo and high blood pressure had proved very valuable, namely, glonoin.

The members of the profession in this country were certainly very fortunate in having Mr. Lake come to us and give us the result of his wide experience in labyrinthine work.

Dr. Dench said that he was greatly indebted to Mr. Lake for the classification of the symptom of aural vertigo. He believed this classification was the best that had ever been presented to the medical profession. Dr. Dench remarked that he recalled several cases of aural vertigo which had come under his observation about which he would like to speak. In three of these a middle ear lesion was present. One of these patients had been previously examined by a prominent otologist, who had pronounced the case not one of aural vertigo. In all of these cases the vertiginous symptoms were extremely well marked. In two of the patients the attacks of dizziness were so severe as to prevent them from attending to their regular work. One of the patients, when first seen, had such frequent and severe vertiginous attacks, that it was necessary for him to go about with an attendant. In all of these cases the introduction of two drops of a 2 per cent solution of pilocarpin into the tympanic cavity, through the eustachian catheter, was followed by a complete disappearance of the vertiginous attacks, and by great improvement in the hearing. One of these patients had been under occasional

observation for over a year and a half, and had had no vertigo for over one year. All three patients were now attending to their regular occupations without inconvenience.

In another class of cases operations upon the middle ear were indicated and were of distinct value. In one case the malleus, incus and drum membrane had been removed and the oval window opened, although the stapes could not be extracted. This patient had had no vertigo since the operation. The hearing, however, was at present completely lost.

Referring to the suggestion of Mr. Lake, that increased blood pressure may act as a causative factor in these cases, Dr. Dench believed that two factors were always present in disturbed blood pressure in cases of aural vertigo. One was a previous lesion, either within the tympanic cavity or within the labyrinth, and the second was a disturbance of the blood pressure. The lesion of the internal or middle ear would be reevaled by tests of the hearing and of the static labyrinth. Where the blood pressure factor and the aural factors were combined, the relief of either factor would be followed by a cessation of the vertigo. For instance, if the blood pressure is increased, and the aural lesion is slight, such drugs as glonoin or the iodids, which will reduce the blood pressure, might be sufficient to overcome the vertiginous attacks without any aural treatment; or, if the blood pressure were not too far removed from the normal standard, the relief of the aural condition, provided this were in the middle ear, might also relieve the vertiginous attacks, and the blood pressure factor of the vertigo might be disregarded. The same could be said of those cases which are supposed to follow intestinal toxemia. Here Dr. Dench believed that there was always some lesion of the auditory apparatus present which determined the character of the symptoms. The labyrinth being in an unstable condition, as it were, due either to some organic change in the labyrinth itself, or to some disturbance of the labyrinth secondary to some pathologic change in the middle ear, was more easily affected by a toxin than it would be under normal conditions. Hence, the patient had an attack of vertigo as the result of his intestinal toxemia, rather than some other manifestations of the toxic condition.

With reference to specific cases, Dr. Dench reported the case of a patient who was suffering from a very mild middle ear

lesion, involving both ears. This patient had severe attacks of vertigo. The Wassermann reaction was positive, bone conduction was diminished, and the galvanic test showed hyperexcitability of the auditory nerve. This patient had been under antispecific treatment, but the symptoms had not been relieved. He was finally given heroic doses of mercury, and made a complete recovery.

Dr. Dench also called attention to the value of taking the galvanic reactions of the auditory nerve in every case of vertigo, and he believed that a great deal would be learned from a careful study of these reactions.

The speaker said also that he wanted to confirm what Dr. McKernon had noted regarding the internal administration of pilocarpin. No effect could be expected from this drug unless it was administered regularly for a period of from six to eight weeks.

Dr. Philip Hammond, Boston: I am sure we are all indebted to Mr. Lake for the admirable classification of labyrinthine diseases which he has presented this evening. A paper such as we have just listened to is epoch making, in that it deals with a form of vertigo which is but little considered. Much is appearing at the present time in our medical publications concerning the vertigo of labyrinthine irritation, particularly in suppurative cases, but little progress has been made in the study of this affliction in nonsuppurative otitis media. The mere mention of the term aural vertigo generally calls to mind Meniere's disease, and for the average man this is an all comprehensive term. We all know about the original case described by Meniere, and although this name has been fastened on to thousands of similar attacks since then, there have been but about fifteen authentic cases verified at autopsy. Because there are so few cases of vertigo which can properly be called Meniere's disease, and because there are so many patients made dizzy by apoplectiform seizures which in some respects simulate the classical description of this disease, many writers believe with Amberg that it would be better to drop the name Meniere's disease from aural nomenclature, adopting in its stead some more definite term. Therefore, we should be doubly grateful to Mr. Lake for presenting for our consideration examples of other pathologic conditions which are capable of causing vertigo.

As has been intimated in his excellent paper, which we have just heard, progress in our knowledge of this condition is slow, particularly as we have so few cases that can be examined post-mortem with any definite history of the condition while living. It is necessary that we group together the known facts relative to this disease before any attempts at theorizing are possible. It is only within recent years that a discrimination has been made between true Meniere's disease and aural vertigo of apoplectiform type, and it is only recently that increased labyrinthine pressure has been thought to be the cause of such attacks.

I am very glad that Mr. Lake has expressed such decided views on the question of increased pressure as the cause of vertigo. He tells us this evening that in none of the cases on which he has operated has he seen fluid under pressure in the labyrinth. Now this finding has been confirmed by the experience of one of my colleagues in a case which he reported before the International Otological Congress in Boston last summer. I refer to the case operated upon by Dr. Walker. In this case there was some fluid in the labyrinth, but the quantity was so small that it did not even run out after opening the bony capsule. Now this at once leads us to believe that some definite change has taken place in that part of the ear which forms the perilymph, otherwise there would have been in these cases the escape of a quantity of fluid.

That the perilymph must be able to run into the labyrinth freely from some source is shown by the quantities which are secreted following the evulsion of the stapes or accidental opening of the bony labyrinth. So good authorities as Schwalbe and Siebenmann assert that there is no definite connection between the labyrinth and the subarachnoid space. What seems to be highly probable is that there exist small passages, as the aqueductus cochleæ or spaces around the internal auditory meatus, or both, through which fluid may percolate. Because of the conditions found in these cases reported by Mr. Lake, and confirmed by the observations of Walker, it would seem highly probable that in certain cases of otosclerosis these natural fissures through which fluid comes to the labyrinth are more or less, if not absolutely, obstructed. While increased pressure may not be the direct cause of the vertigo, it has been demonstrated that sudden changes in press-

ure do cause it. It has been observed that in practically all cases of aural vertigo there is a certain amount of deafness gradually increasing, due either to otosclerosis or to an adhesive process around the stapes and round window, thereby eliminating one of the normal means for allowing for variations of pressure in the labyrinth.

In order to seek for a means of curing this disease, one must look for the cause of the suddenness of the attacks. As has been shown by Mr. Lake, many of these attacks come on while the patient is absolutely quiet, and in fact in some instances during sleep. In these cases it is very difficult to imagine that the vertigo is caused by a gradual increase in the labyrinthine pressure, but it is very easy to imagine that it is the result of a sudden lessening of that pressure. For instance, in glaucoma there is ample opportunity for expansion of the eye with the increase in pressure of the fluid within it. In the ear, however, we have no such opportunity for expansion and increase in the quantity of the fluid, and there is very definite and very severe pressure brought to bear upon all parts of this delicate mechanism. Now, assuming that we have such an increase in pressure within the labyrinth, one can imagine that after reaching a certain limit something must give way. The labyrinth, as is well known, is surrounded by a bony capsule having no definite apertures leading therefrom.

The first decided step forward in the treatment of aural vertigo was the introduction of lumbar puncture by Babinsky. Moiard agrees with Babinsky in his conclusions that great relief is attained in cases of vertigo following this procedure, the beneficial effects lasting in some cases more than a year. They both state that the symptoms are frequently augumented immediately following the puncture, improvement coming only after a period varying from several days to a week. In his investigations Babinsky ascertained first that a galvanic current passed through the head causes vertigo in normal human beings; second, that with otosclerosis or vertigo there was a greater resistance to the current; and third, that this resistance was reduced following lumbar puncture. Reasoning that the strength of current required to cause vertigo when passed through the vestibule was diminished following lumbar puncture, he thought that without doubt the lessening of the pressure in the spinal canal affected the pressure in the labyrinth.

His conclusions, based on the study of a considerable number of cases, were that vertigo is almost always lessened, that the hearing might not be improved, and that the distressing tinnitus persisted. This work of Babinsky has been more recently confirmed by the investigations of Blake and Putnam in Boston.

Experiments through many years, beginning with the work of Ewald on birds and animals, have shown that the sense of equilibrium is situated in the semicircular canals and vestibule. These studies have been confirmed by the more recent work of Neumann, Bárány and others on the infected labyrinth in the human body. Our most recent knowledge leads us to believe that motion imparted to the hair cells on the crista ampullaris by the endolymph as it moves in the semicircular canals, causes sensations of change in position, and if the vestibule with its ampullæ be destroyed, the ability to perceive changes in position will be lost. In other words, the vestibule is the important thing to destroy, and the semicircular canals are only of secondary importance.

We are indebted to our English colleagues, Milligan and Lake for our knowledge of the most radical procedure in these cases of nonsuppurative labyrinthitis. While this work is intensely interesting and instructive, and it is hoped that much good may come from it, it is certainly not a method that will be employed for the relief of all cases of vertigo, nor is it advocated as such by its originators.

This brings us to a consideration of the means at our command for combating this malady. First and foremost is some method of depletion. This we are accustomed to accomplish in many ways. Pilocarpin subcutaneously, the use of a seton, the administering of large doses of salts, and the ingestion of adrenal extract, as suggested by Randall, are among those in use. If these fail to relieve, the advisability of lumbar puncture should certainly be considered.

Finally, should we resort to this radical operation on the labyrinth, the subsequent history should be most carefully followed, as we must remember that almost equally good results are attendant upon lumbar puncture—but they are not permanent. Opening the labyrinth would be but a more direct way of reducing intralabyrinthine pressure, were we to stop at the simple opening. The whole subject is one of most absorbing

interest, and promises to open up new fields for endeavor in our special work.

Dr. Percy Fridenberg: We have to congratulate ourselves and to thank Mr. Lake for a careful, practical study of an abstruse symptom complex, vertigo, from the clinical point of view, which, as he has pointed out, gives us the firm basis of observed fact from which to start on our deductions, and which is the only really promising one, at least in our present knowledge, or lack of it, regarding the pathology and much of the physiology of equilibration. We have become accustomed to accepting without question certain conclusions which are but little more than inferences. This applies, for instance, to the etiologic factor, so freely accused, of arteriosclerosis. We know that arteriosclerotics often have vertigo, or rather get dizzy, but so do the neurasthenic and the hysterical. What is the underlying structural or functional change? If it is anemia secondary to narrowing of arteries and slowed blood current, why is the vertigo not continuous? Why do we not get severe vertigo in other blood states characterized by insufficient circulation or nourishment? There is no doubt that circulatory disturbances play an important role in the causation of vertigo, and that hyperemia and congestion, acute or chronic, may be as deleterious as the opposite condition. I need merely refer to the vertiginous states induced by nitroglycerin and similar vasodilators, the vertigo of acute alcohol intoxication, and the dizziness produced in plethoric subjects by sudden or prolonged stooping. That circulatory conditions, too, are of clinical importance in true labyrinthine vertigo, without actual suppuration and pressure from exudate, seems to me at least probable. Hyperemia of the internal ear or serous labyrinthitis must be considered. I believe that we may even have a transient condition of hyperesthesia of the labyrinth without morbid changes, due to toxins or transitory irritation or vascular circulatory anomalies, and this I should like to call labyrinthism rather than labyrinthitis, just as we speak of meningism. I doubt whether an actual pathologic contraction of either the semicircular canals or of the other tubes mentioned by the reader could be held responsible for a transient or intermittent vertigo. It could of course explain a continuous symptom of this sort, but it would be difficult to understand how the vertigo could be relieved spontaneously as long as the narrowing, if that

really was the cause, remained, as it would have to do on a permanent histologic basis. Among the factors of a reflex labyrinthism or of a vertiginous state we must consider eye strain, especially the variety dependent on imbalance of the ocular muscles. When there is actual diplopia, as in ocular palsy, the vertigo may be so extreme as to cause nausea and vomiting and require the exclusion of the affected eye from the visual act.

In the individual case we may have to decide whether the coexistence of deafness with vertigo or of specific disease with vertigo indicates a casual relation or merely a fortuitous coincidence, and this question is by no means academic. Considering our limited knowledge of the pathology and minute physiology of the inner ear, we may only be able to arrive at a decision ex juvantibus.

In connection with specific disease, I wish to refer to salvarsan injections, and to the fact that attacks of vertigo with other marked systemic disturbance are not rare. Various theories have been advanced, but we are still in doubt whether we have to deal with acute toxic (arsenical) vestibular neuritis, labyrinthine hyperemia due to setting free of a certain number of spirochetæ in the blood stream, or a Herxheimer's reaction.

The arbitrary age limit of forty by no means excludes arteriosclerosis, at least in America. This fact might explain the variable result of operative treatment and the necessity of making two subdivisions of classes, those benefited, and those unimproved. The latter were possibly juvenile arteriosclerotics, or at least premature presenile arteriosclerotics. We may in time to come realize that there are other changes, possibly in nerves and end organs, as well as in bony tissues and structures, quite analogous to arteriosclerosis in their deleterious effect on function, and that such morbid processes, like certain low grade intoxications and infections, may well produce a gradual and slowly progressive deafness, together with nutritional and circulatory disturbances, giving rise to vertigo and vertiginous states. Gastrointestinal or renal autointoxication may produce a symptom complex closely simulating brain tumor, with nausea and vomiting in the foreground of the clinical picture. How much of this is due to toxic causes and how much to increased intracranial pressure? We know, since Cushing has pointed it out to us, that the choked disc of

Bright's neuroretinitis is not due only or even mainly to toxic and vascular anomalies, as was accepted traditionally, but is a manifestation of high pressure within the skull. Similar factors acting in less intense degrees would undoubtedly produce vertigo.

As to labyrinthine hemorrhage, it seems probable that this may occur at a comparatively early stage in life. While it is true that the hemorrhages due to pronounced atheroma are characteristic of senility, we may, I believe, have presenile changes, just as we have them in the retina, where, too, as I may remind you, it was formerly thought that hemorrhages were only found in old age. Various blood states, leukemia, malaria, pernicious anemia, purpura, septic systemic and local infections, and finally some toxic drugs and mineral poisons, predispose to, and excite, capillary hemorrhages in comparatively young subjects.

Tinnitus might be explained as a symptom of autogenous irritation, depending on paralytic dilatation of blood vessels in the cochlea following complete obstruction, as in partial thrombosis or transient spasm. This is quite analogous, clinically and pathologically, to visual symptoms of flashes of light observed in cases of vascular spasm of the retina and in the dazzling scotoma of hemicrania, which is undoubtedly due to localized brain anemia.

Among traumatic agents we must consider the effect of sudden and intense stimulation by sound. It would seem not at all improbable that very high pitched or penetrating tones might cause such vibration as to tear a number of delicate vessels in cochlea or labyrinth. I have known the low pitched vibrating hum of a locomotive exhaust to produce most annoying symptoms of confusion very much like vertigo.

Persistence of equilibrational disturbances, as inability to walk in the dark, after the cessation of tinnitis, would seem to indicate that neuritis of the vestibular nerve had progressed to degeneration and complete loss of function, which was then to be compensated, in time, by the contralateral labyrinth.

Repeated and sustained emotional excitement may produce effects not unlike those of arteriosclerosis, of which it is again a frequent cause. Vertigo in the morning is not infrequently observed as a result of sexual erethism, continued and unrelieved by the natural climax and consequent relief of tension,

physical, vascular, and emotional, and in such other emotional stress as continued apprehension of loss of money, or of reputation, worry, anger.

We must bear in mind the possible influence of toxic factors, especially in gouty subjects and in some of the cases with diminished blood pressure.

Vertigo with ocular symptoms borders on the migraine complex, which, as we know to our regret, is not at all uncommon. The account of the case of visual aura followed by intense vertigo and nausea certainly suggests eye strain with scintillating scotoma. Vertigo coming on with bright light, as reported, or, and this is curious, with loud noises, suggests retinocerebral vascular spasm, or the peculiar synergy known as photophonism or phonophotism, if you prefer, noted in not a few subjects of unstable emotional equilibrium. With these subjects every perception of sound produces a sensation of color, and vice versa; they "hear" red, green, blue, as the case may be, with the tones of different instruments; the violin is green, the clarinet yellow, and so on.

A visual or rather an ocular component may be present in a number of forms of what we may term reflex vertigo. Thus in the vertigo of seasickness, it is hard to say just how much is due to the constant changes in position of surrounding objects, how much to splanchnic irritation, how much to actual rotation involving not only the semicircular canals but the entire brain and meninges. The effect of constrained eye positions and abnormal muscle action, intensified by eye strain and retinal torpor due to insufficient illumination, is seen in miner's nystagmus, which is often associated with what must be a purely reflex vertigo. Even lesser degrees of muscular imblance may, as has already been stated, cause similar vertiginous states. It imposes on us the responsibility of having a careful and complete ophthalmoscopic examination made of such cases as are not evidently aural, and even in these we may, as I have done, make the interesting and rather surprising discovery of latent and unsuspected eye strain.

Dr. T. P. Berens said that he only wished to emphasize what had been said about the danger of operations on the labyrinth, and to thank Mr. Lake for coming over and presenting this paper, for the excellence of the classification of

these cases, and for the unusual clearness with which the subject had been treated.

MR. LAKE, closing the discussion, alluded first to the very clear and interesting remarks made by Dr. Dana, especially upon the influence of the cerebellum upon vertigo.

Dealing then with the criticisms which had been made, as a whole. The first point was the brief allusion he had made to specific disease—lues. This was chiefly because he had omitted many parts of his original paper on account of its length, and specific disease was one. In considering the value of salvarsan and its dangers, Mr. Lake took the opportunity of expressing his astonishment that so powerful a drug should be given intravenously with no precautions and in a standard dose—not considering the body weight, nor the state of the blood, nor its coagulability. As to the intratympanic use of pilocarpin, Mr. Dench was much to be congratulated upon his results.

With regard again to the galvanic tests and reactions in cases of aural vertigo, Mr. Lake said that time had been wanting, and he thought would always be wanting, to allow of all these tests being employed. For time represents the doctor's capital.

Mr. Lake had never regarded Babinsky's work as quite reliable from an aurist's point of view, the absence of tests and proper aural treatment rendering them useless. At all events, the effect of lumbar puncture was, inter alia, to lower the blood pressure, an effect to be obtained otherwise.

With regard to increased labyrinthine pressure, personally he was skeptical as to its existence.

In respect to operation, he had removed all the semicircular canals once, but the shock was very severe and so he proceeded to attack the vestibule. As for the operation being dangerous or not, he believed that turned, as he has hinted, upon the question of strict antisepsis.

In conclusion, Mr. Lake expressed his great appreciation of the honor done him, not alone by the invitation he had received from their chairman, but by the attendance of so many of his colleagues, especially to those who had covered long distances for so short a time.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of November 19, 1912.

DR. JOSEPH C. BECK, PRESIDENT.

PRESENTATION OF CASES.

Sarcoma of the Tonsil or Tonsillar Manifestation of Hodgkins' Disease.

DR. G. A. TORRISON: E. S., 68 years old, engineer, had been troubled for the past week with difficult and painful deglutition. He had entered the Presbyterian Hospital a year ago, where a diagnosis of Hodgkins' disease had been made. Family history was negative. He denied having had syphilis. Inspection of the throat showed an enormously enlarged left tonsil, which reached nearly over to its fellow on the opposite side. The latter was normal in size. There were enlarged glands in the left side of the neck. The surface of the enlarged tonsil was smooth and presented a reddened and glazed appearance, suggesting a large tonsillar abscess. An incision gave no pus, the knife entering a cavity which proved to be an enlarged crypt. The probable diagnosis then lay between sarcoma and a tonsillar manifestation of Hodgkins' disease, the latter being the more likely.

Perforation of Shrapnell's Membrane.

DR. J. HOLINGER: The patient was a painter, 32 years of age. He has been suffering from acute otitis media for the last five days, with very copious discharge. There was a tit-shaped perforation in the region of Shrapnell's membrane. The perforation of Shrapnell's membrane is always due to a chronic process, says Bezold. Politzer, on the other hand, insists that he saw perforations in this part in acute inflammations. This case seemed to be an argument in favor of Politzer's theory. Dr. Holinger slit the tit, and today, two weeks after the demonstration, the tit had disappeared and the perforation was

in the posterior upper quadrant. The swelling and the pus must have moved the soft parts. So the case supports Bezold's contention.

Tuberculosis of the Nasal Cavities.

DR. OTIS H. MACLAY presented a case of suspected tubercular destruction of the nasal cavities, the turbinate tissues entirely gone, much crusting, some odor, bony surfaces exposed in various regions of the nose. Antisyphilitic treatment had been tried without apparent benefit; for the last two months has been under injections of tuberculin, with decided improvement.

Paper: Resonators, With Special Reference to the Schaefer Apparatus.*

DR. ROBERT SONNENSCHEIN: Resonators are appliances which have the property of selecting out and reinforcing certain sounds. Resonance is the reinforcement or intensification of sounds due to the union of direct and reflected waves. Resonators have been used in various ways by different men, such as Helmholtz, Schaefer, Abraham, Wien, Wastzmann, and others, for the investigation of many physical problems, among them the analysis of tones, especially the vowels, the study of "interference tones," etc. Although very simple apparatus, such as a lamp chimney, a tube of cardboard, or a bottle, may serve as a resonator, still the best thing is either a spherical resonator or a metal cylindrical tube, both of which may be connected with the ear. If open at both ends, the pitch of a resonator is higher than if one end is closed. On the other hand, a resonator is of deeper pitch (a) the larger the air containing space, and (b) the narrower the sound opening happens to be. Tones of medium pitch are the ones most intensely reinforced, the very low and the very high ones much less so.

The Schaefer set of resonators consists of four cylindrical brass tubes of different lengths, but the same diameter. One end is open, the other closed by a plate having a central aperture. Over the latter is a conical extension intended to be placed in the meatus auditorius, or to be connected with tubing for introduction into the ear. For occluding the small opening when the resonators are not to be inserted into the meatus, rubber plugs are provided. Each resonator tube fits accurately into another cylinder, but can be moved in and out. The inner

tube is graduated in millimeters, and at the same time the tones for which the resonator acts at different lengths are indicated by letters giving the notes of the scale. The gradation corresponds to the tones of the temperate scale based on a^1 (435 v.d.), at a temperature of 18 degrees C. (about 64.4 degrees F.).

The advantage of the Schaefer resonators seems to lie in their being compact, accurate, comparatively cheap, and easily adjustable to any desired tone between A (108 v. d.) and c^3 (1024 v.d.).

CONCLUSIONS.

Resonators can be used for various purposes: (1) In physical research, in the analysis of tones, etc.; (2) in testing tuning forks to see if the actual tone is the one claimed to be present; (3) for determining the pitch of unmarked forks or that of any other sounding body whose tone comes within the range of the resonators employed; (4) and last, but not least, for testing the absolute duration of hearing for any tone or set of sounds.

DISCUSSION.

DR. RICHARD H. BROWN asked whether, if you get a resonator tuned to about a medium pitch, such as a man talking in a very monotonous voice, that can be used by a person with diminished hearing, to catch enough of the sound vibrations to enable that person to hear better? He wondered if that had been tried.

DR. J. HOLINGER did not believe that resonators have been used or tried for the purpose spoken of by Dr. Brown, and he did not believe that it would be feasible. The bulk of the resonators excluded their being carried around by a person. One important application, however, might be this: The resonators of the well known span from c^1 to g^1 might be used in determining the degree of defect in hearing in children, in order to find out which deafmutes could be educated through their sense of hearing. If hearing extended over this well known span and amounted to hearing the tuning forks of this pitch for ten per cent of their normal duration, hearing could be used for educating children.

DR. EDWIN PYNCHON asked Dr. Sonnenschein if he had had any experience with the audiometer. This instrument had a little bell, the tone of which could be modified as desired by the examiner.

DR. SONNENSCHEIN, in closing the discussion, added a little to what Dr. Holinger had said in answering Dr. Brown's question. It would not be possible to use these resonators very well as an aid to hearing, for the reason that, while it is true that the range not only covers the range given by Bezold, that is, from b^1 to g^2 (these go considerably lower than that—an octave, and also a full octave higher), we must remember that it is the overtones in the various vowels that give the quality to the voice. Now, even though people speak in a socalled monotone, every vowel has certain overtones, and each resonator will resound only to one particular tone when adjusted at a certain length.

So far as teaching deafmutes was concerned, which was referred to by Dr. Holinger, of course that is very important, but he thinks that the same remarks would apply, namely, that these resonators not only cover this area, but more than that. Regarding Dr. Pynchon's question about the audiometer, Dr. Sonnenschein has had no practical experience with it. It is an apparatus not for reinforcing sound, but for testing hearing by means of a hammer enclosed in a sounding box, which is controlled entirely from the outside, so that the patient has no way of judging what intensity of sound is being produced. This is connected with the ear by tubing as in a stethoscope. One can produce an intensity of sound from 0 to 10, and thus see when the patient begins to appreciate that sound.

DR. BROWN asked whether it is known what the actual difference of pitch is in the vowel tones in monotone talk.

DR. SONNENSCHEIN replied that even in a monotone the quality of speech depends entirely on the articulation and emphasis, whereas the specific tone would have to have the same pitch. Even if a person speaks in a monotone and pronounces different vowels, they are of different pitch.

DR. BROWN asked what is the difference.

DR. SONNENSCHEIN said he has forgotten the exact difference, but believes that Wolf showed that there is a range of almost three octaves in pitch of the various vowels and consonants.

Paper: The Physiology of the Semicircular Canals—A Lantern Demonstration.

By George E. Shambaugh, M. D.,

Chicago.

Dr. Shambaugh pointed out the importance of obtaining a correct idea of the anatomy of the end organ in the semicircular canal, and the difficulties encountered in making preparations of these end organs. The end organ in the semicircular canals is the crista ampullaris, which forms a ridge placed so as to receive the full impact of the endolymph passing from the utricle into the canal or from the canal into the utricle. Both sides and the crest of the crista are covered by hair cells. Superimposed above the crista is placed the cupola, which is separated by an appreciable space from the free surface of the epithelium. The hairs of the hair cells project into the under surface of the cupola. The cupola is a stationary cap which is not capable of being displaced by endolymph currents.

ANALYSIS OF THE PHYSICAL REACTIONS WHICH PRODUCE A STIMULATION OF THE HAIR CELLS OF THE CRISTA AMPULLARIS.

1. The stimulation of the hair cells is the result of an irritation applied to the projecting hairs.

2. The irritation of the hairs is brought about by an interaction between the cupola and the hairs.

3. This interaction is occasioned normally by the impaction of endolymph currents against the sides of the cupola.

4. Only those hair cells on the side of the crista receiving the impact are stimulated by a current of endolymph.

Conclusions that are still unsettled in the analysis of these physical reactions are:

1. Is the duration of the stimulation of the hair cells the same as the duration of the endolymph current?

2. Is the duration of the nystagmus the same as the duration of the endolymph?

Since we are able to observe objectively the duration of nystagmus, the answer to these questions can be approached by finding some way of estimating the duration of an endolymph current. Three methods are known by which the duration of

the endolymph current can be estimated: (1) The Ewald experiment; (2) the compression test in fistula cases, and (3) the fact that in caloric stimulation of the semicircular canal it is necessary, in order to get an endolymph current, that the canal be placed in a vertical position. The endolymph current which is occasioned in this way by caloric stimulation ceases the moment the canal is placed in the horizontal plane. In all three of these experiments we observe that the duration of the nystagmus produced by an endolymph current stops apparently the moment the endolymph current ceases.

PHENOMENA IN ROTATION EXPERIMENT.

On starting rotation a patient develops a nystagmus in the direction of the rotation. After rotation has continued for some moments this nystagmus disappears. If the speed of rotation is now accelerated, this nystagmus will again return. On suddenly stopping rotation a nystagmus develops in the opposite direction, which also lasts for a number of seconds before subsiding. The simplest explanation for these phenomena was offered by Breuer and Crum Brown, who explained them as the result of inertia of the endolymph. Breuer gave up the idea later, because of the difficulty in accounting for the inertia of endolymph in the small canals producing an endolymph current that would last long enough to explain the duration of the turning and after-turning nystagmus. Breuer accepted the conclusions that both on starting and on stopping turning there can be but a momentary impulse to the endolymph. He still believed that the duration of the nystagmus depended upon the duration of the stimulation of the hair cells. In order, therefore, to account for the stimulation of the hair cells lasting long enough to explain the duration of nystagmus in the rotation tests, Breuer assumes that the momentary impaction of endolymph displaces the cupola. This displacement of the cupola produces a stimulation of the hair cells that continues until the cupola has been drawn back to its normal position. Bárány's explanation for the rotation phenomena is that there are two nystagmus centers, each containing stored energy which requires just so much time to become expended after stimulation. One center produces nystagmus toward the right, the other toward the left. Bárány believes that there is

but a momentary impulse to the endolymph on starting and on stopping rotation. This produces a momentary stimulation of the hair cells. The impulse on starting rotation stimulates the center which produces nystagmus toward one side, and the impulse on stopping rotation produces stimulation of the center which directs nystagmus toward the other side.

Fundamental objections to both the Breuer and Bárány hypothesis are the experiments cited above, where in the fistula test and in the caloric stimulation the nystagmus stops the moment the endolymph current stops. If either the Breuer or the Bárány hypothesis were correct, the first impact of endolymph in both of these tests should produce a nystagmus which would continue for some moments after the endolymph current ceased.

Other phenomena in the rotation experiment are that the maximum after-nystagmus is obtained after about ten rotations, and that a shorter after-nystagmus is obtained when we rotate a patient a shorter or a longer time; and the fact that after a prolonged rotation in certain individuals there is not only a shorter after-nystagmus, but when this dies out there develops a nystagmus directed toward the same side as the turning nystagmus, a socalled after after-nystagmus. These phenomena are not accounted for in Breuer's theory, nor can they be explained with Bárány's hypothesis. They are explained, however, as phenomena of fatigue from overstimulation, if we accept the theory that the duration of the nystagmus is dependent upon the duration of an endolymph current.

ORIGIN OF LABYRINTH TONUS.

The phenomena of labyrinth tonus can be intelligently understood only by keeping in mind the following facts in the physiology of the semicircular canals: (1) An endolymph current stimulates only the hair cells on the side of the crista receiving the impact; (2) an endolymph current in one direction in a canal produces stimulation of the muscles which direct nystagmus toward one side; an endolymph current in the opposite direction stimulates the muscles which direct nystagmus toward the other side. In each semicircular canal the stimulation which results from an endolymph current which directs nystagmus toward the same side is greater than the stimulation from the endolymph current which directs nystagmus to the

other side. In other words, in each crista impulses from the hair cells on one side produce nystagmus in one direction, and impulses from the hair cells on the other side produce a nystagmus in the opposite direction, the stronger impulses coming from those hair cells which direct nystagmus toward the same side. Labyrinth tonus is the result of impulses constantly emanating from the hair cells on both sides of each crista. From each labyrinth, therefore, tonus impulses emanate which tend to produce nystagmus toward both sides; but since the stronger impulses are those which produce nystagmus toward the same side, the tonus from a labyrinth, if unchecked by corresponding impulses from the opposite labyrinth, will always produce nystagmus toward the same side. The stimulation of the hair cells which produces tonus impulses is probably occasioned by the intralabyrinth pulsations associated with each beat of the heart. Any unilateral increase in the pulsations would tend to increase the tonus from a labyrinth which would disturb the normal equilibrium and produce a nystagmus directed toward the same side. This condition is observed clinically in cases of labyrinth congestion occasionally associated with acute otitis media; also in most cases of serous labyrinthitis where the congestion of the labyrinth appears to be the most important change. In the severer types of serous labyrinthitis it has been observed that during the onset and during the later stages of recovery there is a spontaneous nystagmus directed toward the same side, produced apparently by hyperemia in the labyrinth. During the height of the process, when the serous exudate is sufficient to paralyze the action of the hair cells, the labyrinth tonus in this labyrinth is suppressed, and for a time spontaneous nystagmus is directed toward the normal side.

In cases of diffuse suppuration of the labyrinth the spontaneous nystagmus is directed toward the normal side, because now the normal tonus from this labyrinth is unchecked by impulses from the diseased labyrinth.

ORIGIN OF COMPENSATORY TONUS AFTER DESTRUCTION OF THE LABYRINTH.

The disturbance of equilibrium which follows the suppression of labyrinth tonus when one labyrinth is destroyed, rapidly disappears because compensatory tonus develops to take the

place of the tonus from the destroyed labyrinth. Tonus impulses to the voluntary muscles have two sources: (1) Tonus from the labyrinth, and (2) tonus from extralabyrinth afferent impulses. The sudden destruction of a labyrinth destroys permanently the labyrinth tonus from this side, and at the same time apparently suppresses temporarily much of the extralabyrinth tonus to the same muscles. In the recovery from the disturbed equilibrium which follows the sudden destruction of a labyrinth, the first process is the return of the extralabyrinth tonus impulses which the sudden shock temporarily suppressed. The next step is a compensatory increase in the extralabyrinth tonus to balance the tonus from the normal labyrinth, and, finally, in this readjustment there often appears to be a compensatory increase in those impulses from the normal labyrinth which tend to produce nystagmus toward the opposite side. It is only in cases of long standing labyrinth destruction, where the tonus impulses from the normal labyrinth which direct nystagmus toward the opposite side apparently increase so as to balance tonus impulses which direct nystagmus toward the same side; in other words, when the impulses from the hair cells on the two sides of the cristæ are equal. When the tonus impulses from the hair cells on both sides of a crista are equal, then the after-nystagmus in the rotation tests will be as strong toward the defective as toward the normal side. This has only been observed in cases of long standing unilateral labyrinth destruction.

DISCUSSION.

Dr. Richard H. Brown believes all of the explanations he has heard, and, so far as he followed Dr. Shambaugh, his explanation, also, presupposes that in rotary nystagmus we have a motion circulation in the canal—take the horizontal canal as an example. If this canal were commensurate with the head, it would be a circle of some four inches in diameter, revolving around its center, but in fact this canal is a little thing of about one-quarter inch or less, and revolving at the end of a radius of some two inches. It seems to him that, revolving in that way, we would have a centrifugal action, the fluid tending to force itself to the outermost circumference and circulating through all of the different canals to the outer part. He would suggest that if some ingenious man could make a pair of glass

labyrinths, enlarged, and fill them with fluid containing a floating pigment, then mount them on a bar of perhaps four feet in length, in exactly the same relation, and rotate them, as in a centrifuge, and then stop them, he could observe the actual play of the pigment in the glass index. Thus far the theories advanced have never seemed convincing to him.

DR. E. R. LEWIS, of Dubuque, Iowa, believes all the members present were greatly in Dr. Shambaugh's debt for the presentation of this very admirable discussion. He wished particularly to say one thing. When Dr. Shambaugh presented this matter some year and a half ago, he could not accept as plausible his idea that nystagmus in one direction was caused by the influence of the cells on one side of the crista, and nystagmus in the other direction was caused by the influence of the cells of the other side of the crista. At that time the doctor did not bring out the point that in this conception the cells on one side exceed in their impulse value the cells on the other side; and the objection which he raised at that time, that this theory was untenable when we consider that nystagmus is produced by the influence of the cathode which stimulates all the cells equally at the same time, was, he thinks, valid. But now that Dr. Shambaugh has explained that he considers the cells, for instance, in the anterior vertical canal on the utricular side, of higher value than the cells on the canal side, and the cells on the canal side of the horizontal of higher value than those of the utricular side, the objection which he raised is satisfactorily disposed of. He thinks Dr. Shambaugh's theory not only plausible, but very acceptable.

Regarding what the essayist said about the influence of the extralabyrinthine tonus in comparison with the influence of labyrinthine tonus, he thinks there is a great deal to suggest that the relative value of the extralabyrinthine tonus is very great. Consider, for instance, that while awake, whether in sitting or standing still, the opposing groups of skeletal muscles are evenly balanced in their contractions, by reason of the tonogenetic current from the afferent impulses, but when we commence to lose consciousness we commence to fail to perceive afferent impulses and, coincidentally, our muscle tonus begins to diminish. With the onset of sleep in sitting position, for example, which gives the pull of gravity a chance to exert its influence upon the upright head, the first evidence of

that diminution of tonus is the nod. The muscles which have held the head upright unconsciously, by reason of that muscle tonus, are relaxed. It cannot be due to labyrinthine tonogenesis alone, because in the very nature of things labyrinthine tonus impulses must go on while asleep as well as awake. It has to do, therefore, with apperception, and if tonogenesis were overwhelmingly of labyrinthine origin, that is, if the amount of tonus impulses emanating from the labyrinth overwhelmingly outmeasured those emanating from other extralabyrinthine sources, we would not find muscle tonus beginning to diminish when we commence to lose consciousness, and atony commensurate with and coincidental with unconsciousness.

Dr. George W. Boot said there is one law in nystagmus which he has never seen mentioned, and which will help in remembering the directions, namely: Nystagmus is always in the opposite direction to the current in the endolymph. While he thinks Dr. Shambaugh is undoubtedly right with reference to tonus from the labyrinth, namely, that the pulsations of the heart give rise to the greater amount of tonus, he believes that a great deal results from sound waves. The semicircular canals are in such intimate relation with the auditory portion of the labyrinth that sound waves which stimulate the cochlea are almost certain to stimulate to some extent the end organs of the semicircular canals. A good illustration of this tonus is in the action of our muscles when some unexpected noise occurs; for instance, when writing, if an unexpected noise occurs, there is a break in the writing. It is the same phenomenon which makes you hold your breath when you see a person walking in a particularly dangerous place. You do not make a sound, because if you did the sound waves would increase the tonus of his muscles, and the greater contraction of the stronger muscles would disturb his balance and cause your friend to fall.

Dr. J. Gordon Wilson was sure that the members all agreed in acknowledging their indebtedness to Dr. Shambaugh for the interesting discussion and the way in which he placed before them some of the problems involved in the physiology of the labyrinth. His only regret is that the essayist tried to give too much. The subject bristles with difficulties which one would like to discuss at length. Many questions suggest themselves.

For instance, the one raised by Dr. Boot—the relation of hearing to the tonus labyrinth. There is no doubt at all, in Dr. Wilson's mind, of this relationship. He has frequently observed in animals, after one labyrinth had been destroyed, that stimulation of the other ear by sound waves would immediately cause that animal to show a reaction indicating stimulation of the labyrinth. We are all agreed that the labyrinth is one of the most important peripheral organs for tonus, but at present he finds great difficulty in accepting Dr. Shambaugh's hypothesis regarding its origin. In discussing tonus it ought to be made very clear that we know tonus chiefly through its increase or diminution; and that of the tonus which exists when the body is at rest, the continuous tonic reflexes, our knowledge is very indefinite. We know that mechanical irritation of the labyrinth and of its nerves will produce alterations of tonus. Also, from observations on the muscles—especially the eye muscles—we must conclude that when the body is at rest there is also an influence constantly going from labyrinth to these eye muscles. But where does this tonus originate, and how is it caused? Brauer believes it originates in connection with the otoliths. Ewald believes it is produced by cilia movements. There is also the physical pressure theory, under which head one may class such an hypothesis as that of Sydney Scott, who looks for an explanation in the heart's action. To all these, serious objections can be urged, which it would take too long to discuss. At present we must conclude that as to how and where this permanent reflex is produced we have not sufficient knowledge.

Dr. Shambaugh suggests that the hair cells play an important part in the production of tonus by pull or by pressure. We do know that in the horizontal canal of birds, movements of endolymph towards the ampulla cause movements opposite to and greater than those from ampulla to canal. But this exists only for the horizontal canal, and is the reverse in the superior. We would require to suppose, on Dr. Shambaugh's hypothesis, that reversal has occurred in the structure of the ampullæ in the two canals, of which we have no atatomic knowledge. An easier explanation would be an hypothesis in regard to the nerve distribution to these parts along lines he has indicated elsewhere.

Dr. Wilson cannot agree with Dr. Shambaugh that the com-

pensatory action of the tonus comes from the other ear. Rotation after one side labyrinth destruction speaks against such a hypothesis. In animals—for instance, the dog—destruction of the labyrinth results in definite torsion of the head, which is permanent, though the animal recovers from the destruction nystagmus in two or three days. So there are certain muscular actions for which he never compensates, and some for which he does. Now, if the other labyrinth is destroyed, within a short time the head readjusts itself. Here the second labyrinth has not compensated, but may have played some part in preventing the compensation. In such cases he cannot see how the phenomena are to be accounted for by a compensatory mechanism lying in the semicircular canals. On the other hand, positive evidence pointing to the cerebrum as being the seat of the compensation has been given in a recent paper, by Wilson and Pike (The Effects of Stimulation and Extirpation of the Labyrinth of the Ear and Their Relation to the Motor System), read before the Royal Society of London in June, 1912, and published in their Transactions.

There are one or two points in all discussions on the physiology of the labyrinth that ought to be clearly understood. One is in regard to the socalled currents of the endolymph. It is unfortunate that the word current is used for movements of the endolymph in the semicircular canals. Currents are unthinkable. What probably occurs is an alteration in pressure. This view finds expression in the now generally accepted Brown-Mach-Brauer hypothesis.

Again, what comes primarily from the labyrinth is not the nystagmus or double movement, but a deviation, which corresponds to the slow phase in the ocular movement. The quick phase in the ocular movement is extralabyrinthine, and one of the points still in dispute is where it originates.

The paper by Dr. Shambaugh was full of information, and its interest increased by the fact that he drew most of his references from man, the ultimate test to which all our experimental work must be applied. In animals we can perform more definite lesions than is possible in man, and, watching their outcome, can arrive at data which we can apply to pathologic conditions in our special work. We strive to bring these experimental and pathologic conditions into agreement. Whether they agree or not, we are ever hopeful that the out-

come will be a clearer conception than we now have of the physiology of the labyrinth.

DR. BOOT said that both Dr. Shambaugh and Dr. Wilson spoke of the greater sensitiveness of the cells on one side of the crista, but did not offer any suggestions as to why this should be. Bárány has given a possible explanation, namely, that the nerve supply on one side of the crista may be greater than on the other.

DR. J. HOLINGER mentioned some experiments published more than ten years ago by Dr. Fischer, in connection with what Dr. Wilson said. Dr. Fischer destroyed one labyrinth completely in a dog. The dog was very sick for a few days, but recovered after about six or seven weeks. Then the other labyrinth was destroyed. After this the dog was sick again, though more seriously, and recovered much more slowly than from the first operation. Then he took other dogs and destroyed a certain part of the cortex of the brain, behind the Rolandic fissure. The dog showed no direct result from this destruction. When, however, in this dog he destroyed one labyrinth, the dog did not recover, and was unable to stand on his feet again. It would have died of inanition if it had not been fed artificially. That would mean that this part of the cortex is capable of taking the place of one labyrinth, because if that part of the cortex was not destroyed the dog would recover. How does that agree with the theory of peripheral tonus?

DR. SHAMBAUGH, in closing, said that Dr. Brown raised the question, whether the centrifugal force might not work to check a motion of the endolymph in the canals, especially since these canals are located at some distance from the pivot of rotation. This question has been brought up before, and he believes there is no difficulty at all in accounting for the motion of the endolymph in a mechanism of this sort. Dr. Brown also suggested the construction of a model to demonstrate exactly what the reactions are in the semicircular canals. This, of course, is the ideal, if it were only possible to construct such a model; but even if it were possible to devise a model that would resemble approximately the semicircular canal mechanism, such a model would be lacking in the finer membranous structures which no doubt influence decidedly the physical reactions resulting from rotation. He cannot see how one could

possibly construct a model that would be of any assistance in this problem. The same question has come up in connection with the action of the membrana tectoria, and it has been suggested that one must devise a model which will demonstrate how the membrana tectoria responds to sound impulses. Of course, it is out of the question to construct such a model, since, in the first place, we don't know exactly what the physical properties of the membrana tectoria are, and, in the second place, even if we did know what these properties are, the mechanism is entirely too delicate and intricate to permit of imitation. Ewald at one time constructed a model in which he placed a taut rubber membrane, which was devised to illustrate the response in the membrana basilaris to the impulses of sound waves. This glass model permitted Ewald to photograph the vibrations of this rubber membrane, as it responded to tones of different pitch. Ewald argued that this illustrated the manner in which the membrana basilaris responds to sound waves. This work of Ewald's attracted a great deal more attention than it deserved. There is no more resemblance between the model constructed by Ewald and the membrana basilaris than between a stretched sheet and strings of a piano.

Dr. Boot and Dr. Wilson suggest that the sound waves entering the ear may pass up through the vestibule and thus bring about a stimulation of the hair cells of the crista, and account in this way for the origin of labyrinth tonus. It would seem that the close anatomic relation between the two parts of the labyrinth might suggest that the same impulses could stimulate the two sets of end-organs. It would be interesting if experiments could be devised that might demonstrate this. He does not see that the observation pointed out by Dr. Boot, that a sharp sound startles a person, is necessarily a proof that this is the result of the increased labyrinth tonus.

Dr. Lewis suggests that extralabyrinth tonus impulses are more important than the tonus from the labyrinth. It would seem a priori that the tonus impulses emanating from a mechanism that has been set aside for the purpose of supplying tonus would be more delicate and more important than the more primitive tonus impulses that emanate from other sources. He does not know of any way by which it is possible to determine whether the labyrinth or the extralabyrinth tonus impulses are the more important.

Dr. Lewis asks whether he believes that the principal function in the semicircular canals is to give tonus? Dr. Shambaugh is inclined to believe that the tonus function of the semicircular canals is as important as any function they may have.

Dr. Lewis thinks that the chief function of this whole apparatus is to sense motion, and that tonus is only a secondary function. He asked Dr. Shambaugh whether he does not believe that the extralabyrinth tonus impulses disappear with loss of consciousness?

Dr. Shambaugh does not see any reason why all the extralabyrinthine tonus impulses must necessarily stop with loss of consciousness, especially if these extralabyrinth tonus impulses have their origin in the action of the viscera, such as the pulsation of the heart and the act of respiration.

Dr. Lewis suggests that the nodding which occurs when one falls asleep in a chair is an evidence that the extralabyrinth tonus disappears with loss of consciousness.

LARYNGOLOGICAL SECTION OF THE ST. LOUIS MEDICAL SOCIETY.

Meeting of January 29, 1913.

Paper: Widening of the Palatal Arch for the Correction of Deflections of the Nasal Septum.*

DR. GEO. V. I. BROWN.—The results of clinical and experimental observation, which were presented for consideration by Dr. Brown, were designed to emphasize the possibility of a very much more extended association between defective nasal and maxillary development, with their consequent pathologic states and influences, and general development, trophic changes, nervous disorders, as well as other more or less remote affections, than is commonly recognized in this relation. Certain features of the etiologic, pathologic and structural considerations which bear upon the influence of these oronasal factors in disease are well understood. Other causal factors, though recognized as important to pathologic manifestations in other regions, are not generally accepted as being of nasal and maxillary origin. A third division must, for the present at least, depend to some extent upon the more or less theoretic application of certain developmental features, connection with which, in the absence of definite knowledge, can be established only by comparative consideration of certain vital phenomena which, viewed in the light of experimental results, clinical manifestations, and the more or less arbitrary application of better understood laws governing bodily growth and metabolism, appear to share at least a measure of interdependence.

To establish unrestricted nasal respiration where the nose is narrow, does not give the necessary breathing space. Dr. Brown practices the rapid separation of the superior maxilla at the median palatine suture, the procedure being accomplished in a period of ten days to two weeks. He stated that clinically we have proven that there is an increase in the size of the nares with corresponding tendency toward improvement in the form of the septum and in the reduction of the

hypertrophic conditions which represent the pathologic results that are the invariable evidence of such malformations, and that these take place with almost mathematical regularity when the upper maxillæ have been forced apart by separation through the median palatine suture without objectionable local or general disturbance. Early treatment of this character can do much to prevent such defects. The nasal processes of the superior maxillary bones are forced apart when these bones are separated by direct pressure across the palate, and they carry with them, to some extent at least, the attached nasal bones. Thus there is an actual increase in the internal measurements of the width of the nose. It also makes possible a separation of the ridges which form a groove for reception of the vomer. This allows the natural resiliency of the septum to take advantage of the additional space thus afforded, with corresponding tendency toward straightening of its curvatures.

In the discussion it was brought out by Dr. Summa and Dr. Lischer that as a whole the orthodontists question the procedure of opening the median maxillary suture. They are of the opinion that the widening of the palatal arch does not result from opening of the suture, but that it is due to the stretching of the thin plate of bone between the maxillary suture and the alveolar process, and that widening of the nasal tract may be due to slight opening of the suture plus stretching of the palatal process of the maxilla. To get the best final result the occlusion of the teeth must be harmonized. The orthodontists take the stand that it is a mistake to carry the upper arch bucally first and then expect an orthodontist to bring the lower in line.